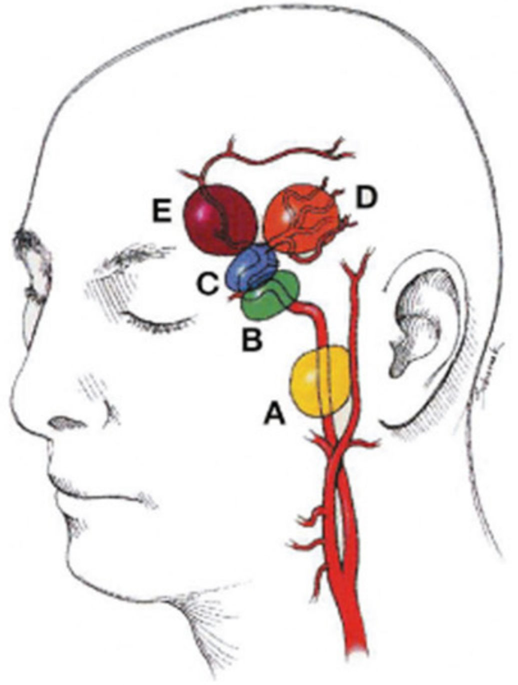
Neurosurgical Operative Atlas

Vascular Neurosurgery

R. Loch Macdonald

Third Edition



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Vascular Neurosurgery

Third Edition

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291 illustrations

Thieme
New York • Stuttgart • Delhi • Rio de Janeiro

Executive Editor: Tim Hiscock
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Printer: King Printing

Library of Congress Cataloging-in-Publication Data

Names: Macdonald, R. Loch (Robert Loch), editor. | American Association of Neurological Surgeons, issuing body.
Title: Neurosurgical operative atlas. Vascular neurosurgery / [edited by] R. Loch Macdonald.
Other titles: Vascular neurosurgery
Description: Third edition. | New York : Thieme, [2019] | Includes bibliographical references and index.
Identifiers: LCCN 2018035616| ISBN 9781626231108 | ISBN 9781604069303 (e-book)
Subjects: | MESH: Vascular Diseases–surgery | Neurosurgical Procedures–methods | Vascular Surgical Procedures–methods | Atlases
Classification: LCC RD594.2 | NLM WG 17 | DDC 617.4/800223–dc23
LC record available at https://lccn.loc.gov/2018035616

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Thieme Publishers New York
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Thieme Publishers Rio de Janeiro, Thieme Publicações Ltda.
Edifício Rodolpho de Paoli, 25º andar
Av. Nilo Peçanha, 50 – Sala 2508
Rio de Janeiro 20020-906 Brasil
+55 21 3172-2297 / +55 21 3172-1896
www.thiemerevinter.com.br

Cover illustration: Barrow Neurological Institute
Typesetting by DiTech Process Solutions

Printed in the United States by King Printing 5 4 3 2 1

ISBN 978-1-62623-110-8

Also available as an e-book:
eISBN 978-1-60406-930-3

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To Sheilah and our three brilliant children, Iain, Robyn, and Erin, and to my parents, Neil and Lea Macdonald.

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Foreword

Loch Macdonald is a very rare, gifted individual who is not only a master cerebrovascular surgeon, but also a true academician and entrepreneur. He has once again created one of the most complete collections of techniques for microneurosurgery. This work is a clear demonstration of his passion and contributions to excellence in operative neurosurgery.

Upon reading the new edition of this book, I am truly impressed by the breadth and depth of the content. The technical pearls are clearly defined and discussed in detail, making this book an excellent resource for residents and fellows as well as for practicing neurosurgeons the night before their surgeries. This collection is a heart-to-heart discussion of "unspoken" technical pearls that make the critical differences in final patient outcomes.

The special features of this offering are the concise nature of the chapters and the instructive accompanying videos. The masters of cerebrovascular surgery are very well represented, and the reader gains an excellent understanding of different styles for managing complex vascular problems.

I wholeheartedly recommend this book to every trainee and practicing neurosurgeon. Again, the leadership of Loch Macdonald in making this contribution possible is sincerely appreciated.

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Preface

It has been 10 years since I edited the last edition of this neurosurgical operative atlas covering vascular neurosurgery. There are three sections: the first covers operations for intracranial aneurysms, the second section addresses brain and spinal vascular malformation, and the third section describes procedures for cerebral ischemia, including the most common bypass (superficial temporal to middle cerebral artery). Some chapters have been added and others describing out-of-date operations have been discarded. Almost all of the chapters have been revised and updated. Each chapter is meant to address the key operative aspects of each of the procedures or diseases that are discussed. This includes patient selection and indications for the procedure, key preoperative tests and information needed to plan the operation, positioning, step-by-step description of the surgery, and the most important postoperative complications to watch for. As many chapters as possible have videos to accompany them to further illustrate the operations. The chapters are mostly short and succinct, meant to be useful for residents, fellows and trainees in neurosurgery as well as practicing neurosurgeons. Beginning residents can review an operation that they will be involved with including its key components and principles. As one progresses through training and then onto practice, the book should be useful to demonstrate methods of other surgeons to make ensure you are current on the latest techniques. Utilizing such a reference is a great way to continue our education as surgeons and remain life-long learners. As some open vascular neurosurgery operations become less common, supplanted by endovascular procedures, this book will be valuable tool for reviewing operative vascular neurosurgery especially as increasingly we may encounter the need to perform a surgery that we may not have done for some time.

Acknowledgments

I am indebted and honored that so many experts in the field of vascular neurosurgery were willing to provide brilliant descriptions of operations that are their area of expertise, and furthermore, for allowing us to include videos of most of the operations. I thank the medical students, residents, fellows, and faculty that I have been able to work with, for their collaboration and support and for teaching me, helping us stay abreast of modern neurosurgery.

Many people from Thieme helped with this project; I especially want to thank Sarah Landis and Kay Conerly.

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3. Discuss the common complications of neurovascular surgical procedures and how to manage them.

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Release and Termination Dates

Original Release Date: 12/1/2018

CME Termination Date: 12/1/2021

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#Educational Content Planners.

Macdonald, Neurosurgical Operative Atlas: Vascular Neurosurgery, 3rd Ed. (ISBN 978-1-62623-110-8),

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1 Vascular and Microsurgical Instrumentation and Equipment

Anthony C. Wang and Jacques J. Morcos

Abstract
Nylen and Holmgren were the first to use a binocular operative microscope, when they began treating otosclerotic hearing loss microsurgically in 1921. Their innovation opened a new chapter in the surgical specialties of neurosurgery, otolaryngology, ophthalmology, and plastic surgery. In 1960, Jules Jacobson coined the term "microsurgery" when he began using a microscope for vascular anastomoses. Enormous advantages in stereoscopic magnification and illumination afforded by use of the operating microscope expanded the range of operable neurosurgical disease by making accessible areas in the brain and spinal cord that were previously unreachable. This chapter provides an overview of the operating microscope and the instruments most commonly used in cerebrovascular neurosurgery. Further, it outlines the principles and techniques that underlie their proper use in the operating room.

Keywords: instrumentation, microsurgery, operating microscope

1.1 Operating Microscope

The microscope serves two main functions—magnification and illumination—permitting smaller exposures, improved visibility for dissection of delicate tissues, and safer hemostasis when working through narrow and deep surgical corridors.[1,2]

Halogen or xenon lamps provide high-intensity illumination along the line of sight. Illumination can be so strong as to cause thermal injury to the tissues, and thus full strength should generally be avoided. Newer generation microscopes tackle this problem by self-adjusting the light intensity.

Surgical microscopes consist of several joints to allow for pan-directional mobility. A key component of the surgical microscope is the mouthpiece switch. This electromagnetic device disengages all the joints along the arm of the microscope with a low-pressure bite, allowing it to float, easily tracing the movements of the head (**Video 1.1**). Mobility along the x-, y-, and z-axes is permitted while maintaining static pitch, yaw, and roll of the microscope head. This allows the surgeon to scan the surgical field, adjust or maintain focus on a target, or focus on a new target while continuing ongoing manual maneuvers. There are many other functions that can be programmed into the various buttons on the microscope hand grips and foot pedal, including neuroimaging for stereotactic guidance, video and still photography, and automatic balancing. Microscope light filters, in conjunction with oral or intravenous dyes, serve as adjuncts in oncologic and vascular neurosurgery. Probably, the most widely used vascular neurosurgery dye is indocyanine green (ICG). ICG video angiography allows visualization of vessels at submillimeter levels and can thus reveal an incompletely clipped aneurysm or an inadvertently stenosed or occluded vessel.[3]

1.2 Operating Microscope Chair

Microsurgery can be lengthy, and muscle fatigue, coupled with mental fatigue, affects even the fittest, most experienced surgeons (**Video 1.1**). The operating chair serves to reduce isometric large muscle activation while operating. In addition to gluteal and lumbar support, proper arm support is particularly crucial. In the starting position, the forearms rest on the distal aspects of the forearm platforms (so that the platforms do not inadvertently strike the table or retractor arms when moving), in a mildly supinated position, such that the hands rest upon the patient on the dorsal aspects of the fifth metacarpal bones. In this manner, the instrument rests on its balance point upon the second proximal phalanx, rather than having the surgeon lift his/her hands and instruments while manipulating the instruments. Hand and arm position is then adjusted according to what the procedure requires.

The height and width of the forearm platforms should be adjusted such that the shoulders and elbows are entirely relaxed, with the elbow joints as close to 90° as the operative field will allow. By neutralizing muscles and joints proximal to the elbows, individual surgical maneuvers are restricted to the wrists and fingers, thereby improving accuracy and endurance.

1.3 Bipolar Electrocautery

Bipolar electrocautery was developed by Malis in the late 1950s by combining a spark-gap transmitter with surgical forceps, the evolution of two-point coagulation as described by Greenwood.[4,5] The bipolar coagulation device allows current to pass between the tips of the forceps, coagulating the tissues in between (albeit with a small degree of lateral thermal spread).[6,7] Should the tips of the bipolar forceps touch, a short circuit is created, and no coagulation occurs. Bipolar cautery is very effective on small blood vessels and around nervous tissue because less current is needed to achieve the same cauterizing effect as with monopolar cautery.

Use of the bipolar forceps on dry tissue can cause char formation and reduce coagulation efficacy, and tissue can stick to the tips of the instrument. In 1972, King and Worpole observed that coagulation could occur even if the tips of the forceps were immersed in irrigation fluid or cerebrospinal fluid (CSF), so they attached an irrigating tube to the bipolar forceps.[8] Though normal saline is the common irrigation solution used, mannitol appears to offer some advantages, since it is nonconductive and the current passes only through the tissue between the forces tips.[9] Bipolar forceps tips coated with Teflon or other materials are available, intended to avoid sticking. Should charring occur, the tips need to be gently cleaned with a wet sponge and not be scraped with sharp instruments, such as scratch pads or scalpels, in order to protect the coating. In addition, the best way to prevent char and sticking is to coagulate in short bursts, constantly opening and closing slightly the tips, without any metal-to-metal contact.

1.4 Bayonetted Instruments and Instrument Length

Bayonetted forceps and angled suction tips are commonly used and are peculiar in that the shafts of the instrument are offset with respect to the axis of the handles, in order to allow the surgeon to look down the barrel without his/her hands blocking the view of the operative field.

The handles of bayonetted instruments are designed to sit between the thumb and the second and third phalanges, and the tips should approximate with gentle pressure. The recoil (opening) force varies according to material and length of the instrument. Forceps with greater recoil force are ideal for tissue dissection and definition of surgical planes. The ideal bayonetted bipolar forceps thus acts as a dual instrument: coagulator and dissector (**Video 1.2**).

When varying lengths of the same instrument are available, the principle of using the shortest possible instrument is rooted in two concepts. The first is to allow the surgeon's hands to rest on the skull. The second is that longer instruments magnify movements (and thus errors) at their tips, proportional to their length. When working on the surface of the brain, for example, for a superficial bypass procedure, short, nonbayonetted instruments are suitable. As dissection is carried deeper, instrument length is increased to allow for the ideal hand position, adding the bayonette configuration after about 6 inches length.

The tips of forceps are designed with variable width and shape. The tips are most commonly straight; however, instruments with curved tips are available, for when the surgeon needs to work around tight corners. When a bipolar forceps is used for electrocautery, usually thinner tips with a smooth inner surface are preferred for more accurate point coagulation. For tissue dissection and grasping, forceps with serrated, toothed, ringed, and/or cupped ends can be used.

Microscissors are available with straight or curved tips that are either sharp or blunt. The length, shape, and directionality of the instrument vary depending on the working surface. Longer, bayonetted microscissors allow for working at a depth (**Video 1.2**). Curved microscissors allow for visualization of the cutting tips with cutting in-line with respect to the surgeon's fingers, and for lifting and cutting simultaneously. Microscissors with straight tips are more easily visualized when cutting in a direction orthogonal to the line of sight.

1.5 Suction Tubes

The appropriate use of the suction tube is crucial for maintaining adequate visualization in two ways. Directed suction is used to maintain an operative field clear from blood, CSF irrigation, and other fluids. At the same time, the appropriately positioned suction shaft, often placed over a patty, provides dynamic brain retraction, thereby minimizing the risk of ischemic injury caused by edges of rigid retractor blades. Neurosurgical suction tubes are available ranging in bore from 3 to 12 French (F), where 3F = 1 mm (▶ Fig. 1.1). The shafts may be rigid or malleable. All microsurgical suction tubes should be connected to flexible in-line extension tubing, rather than directly to the suction vacuum tubing, which is heavy enough to torque the suction in the surgeon's hand inadvertently.

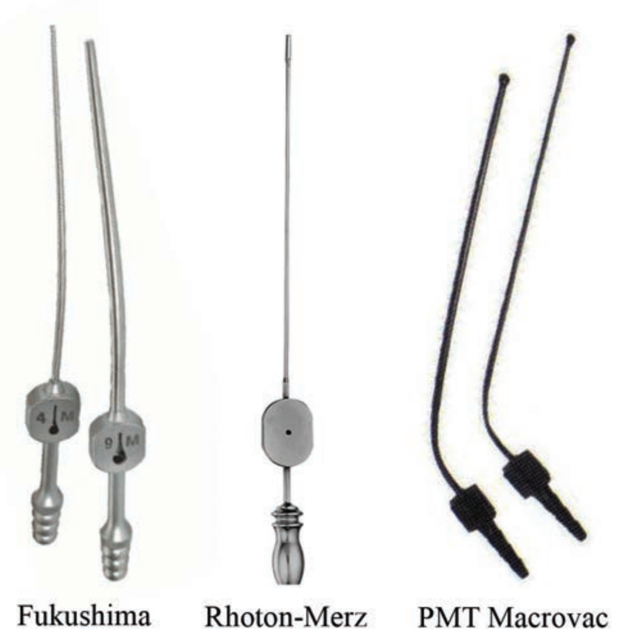
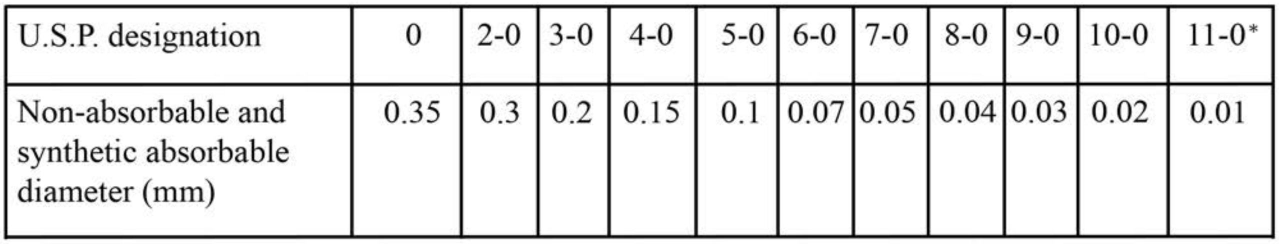


Fig. 1.1 Suction tubes used in cerebrovascular microsurgery. Rhoton-Merz design and PMT MacroVac suction tubes offer a radiused, cylindrical dilation at the tip meant to minimize trauma to adjacent brain when used to retract tissue. The tip of the Fukushima design tapers to a sharp end, and thus is ideal for use where only a slight amount of retraction distal is needed, such as near the basilar apex. The Fukushima suction tube offers a clever tapered design, where the inner diameter is smallest at the tip, such that tissue plugs that can enter the suction tube will not block the suction more proximally. Variable suction intensity on the Fukushima and PMT MacroVac suction tubes is permitted by employing a teardrop-shaped finger vent.

Suction intensity is modulated either by a finger vent, through in-line suction reduction, or at its source. The proximal suction tube is held between the thumb and index finger, such that the surgeon's thumb can roll over the finger vent. The best method for controlling suction is with a slotted vent. Subtle thumb maneuvers modulate suction power and allow continuous rapid control of suction, whereas for suction tips with circular vents the thumb acts as an on/off control. Methods of in-line suction reduction include either a clamp or adapter attached to the vacuum tubing, or extension tubing of reduced bore.

1.6 Vascular Suturing

The approximation of tissues with sutures is essential for primary wound healing. There are many types and sizes of sutures readily available in most operating rooms (▶ Fig. 1.2). They are made of absorbable or nonabsorbable materials, and are either monofilament or comprised of multiple braided filaments. Absorbable sutures are made of animal collagen or synthetic polymers, and material, diameter, and coating influence absorption time. Nonabsorbable sutures are not broken down by hydrolytic or proteolytic enzymatic processes. They are made of natural fibers, synthetic polymers, or metal.



U.S.P. designation	0	2-0	3-0	4-0	5-0	6-0	7-0	8-0	9-0	10-0	11-0*
Non-absorbable and synthetic absorbable diameter (mm)	0.35	0.3	0.2	0.15	0.1	0.07	0.05	0.04	0.03	0.02	0.01

Fig. 1.2 Suture diameter according to United States Pharmacopeia designation. For vascular anastomoses, nonabsorbable, monofilament sutures are necessary. For repair of larger vessels, such as in a carotid endarterectomy, 6–0 polypropylene is a commonly employed suture. For small vessel anastomosis such as STA–MCA or PICA–PICA bypass, a 10–0 or 11–0 polyamide polymer such as nylon is favored. When radial artery or saphenous vein grafts are used for high-flow bypass, 9–0 and 8–0 sutures are typically preferred, respectively.

Polyfilament, or braided, sutures are known to have better handling, flexibility, and tensile strength than their monofilament counterparts. However, they are not suitable for vascular procedures, since they can enlarge suture holes on the vessel edges. Monofilament sutures are smooth and thus slide with less trauma to adjacent tissue.

Due to the lower flexibility of monofilament sutures, a memory effect exists, and thus tying such sutures requires more knots than with braided sutures, and a gentle pause when laying each knot. Careless handling of monofilament sutures may cause focal weakening or breakage of the strand. Surgeons should never hold the suture thread with the jaws of a needle holder, only with the light holding power of a forceps.

All sutures are available with a variety of needles. Needles appropriate for use with microscopic needle holders are curved, ranging from 1/4 to 5/8 of a circle. In microsurgery, 1/4 to 3/8 circle needles are preferred. The needles have a cutting, reverse cutting, or round tapering design. Reverse cutting needles are designed to reduce tearing in penetrating tough tissues such as skin, while tapered needles are used in vascular microsurgery, as they cause the smallest holes. The needle should be grasped at a point one-half to three-fourths the distance from its tip, but not at the connection point with the suture where the needle is hollow, its weakest point.

Castroviejo, a Spanish ophthalmologist, designed a needle holder for use in microsurgery.[10] This instrument has a round or flat handle, and is either straight or curved at its tip. The round shaft allows for smoother, finer rotation between the index finger and the thumb, with the middle finger assisting in supporting the weight (**Video 1.3**).

1.7 Rhoton Microdissectors

The Rhoton microscopic dissector set consists of round canal knives, spatulas, curettes, needle dissectors, and teardrop or ball-tipped dissectors in various sizes and angulations (▶ Fig. 1.3).[11]

The round canal knives are commonly used to dissect tumors from vascular or neural tissue, such as in schwannoma and meningioma surgery. The idea is to hold the instrument in such a way that the sharp edge of the round knife is driven into the plane of separation, while the rounded dorsum simultaneously protects and dissects away the tissue being preserved. These instruments can also be used to dissect arachnoid bands of moderate tenacity.

Every neurosurgeon is familiar with the ubiquitous Rhoton #6 dissector, the narrowest of the spatulas. The spatulas are used to define planes adjacent to vessels, nerves, tumors, arachnoid layers, and other structures in a blunt manner.

The curettes are particularly useful around bone. They offer a sharp edge, which allows the surgeon to delicately dissect dura away from bone, such as in removing the bony shell of an anterior clinoidectomy with a force vector directed away from sensitive structures.

Needle, hook, and teardrop dissectors are offered in varying angulations, ranging from straight to right-angled. Nerve hooks and teardrop dissectors can be used instead of spatulas when extremely fine dissection around particularly sensitive neural and vascular structures is required, such as in separating a vestibular schwannoma from the facial nerve or to define the neck of an aneurysm.

All microdissectors have round handles to allow rolling motion of the instruments between the thumb and the index finger. None employ bayonetted shafts, precisely to prevent movement of the tip through wider arcs. A few microscopic dissecting tools with bayonetted shafts exist, having been designed primarily for endonasal use.

1.8 Vascular Clips and Clip Appliers

The modern crossed-action, helical coiled-spring clip for aneurysmorrhaphy was adapted from an electrician's test clip,[12] though the design was already over a century old at that point. Dipalma reported the use of modified safety pins as simple artery clamps, and aneurysm clips in use today follow the same design.[13] The introduction of the operating microscope necessitated the design of thinner clips and appliers. Yasargil designed thin, crossed-leg spring clips that could apply a strong closing force.[14] Sugita created very long clips, and applied the bayonet shape to both clips and appliers to enhance visibility, inspired by working with Drake.[15] Drake also introduced an innovative round opening (fenestration) at the base of the blades to preserve small vessels and nerves at the base of the aneurysm—specifically, the posterior cerebral arteries while clipping a basilar apex aneurysm.[16]

The production of vascular clips today is regulated by the American Society for Testing and Materials Committee

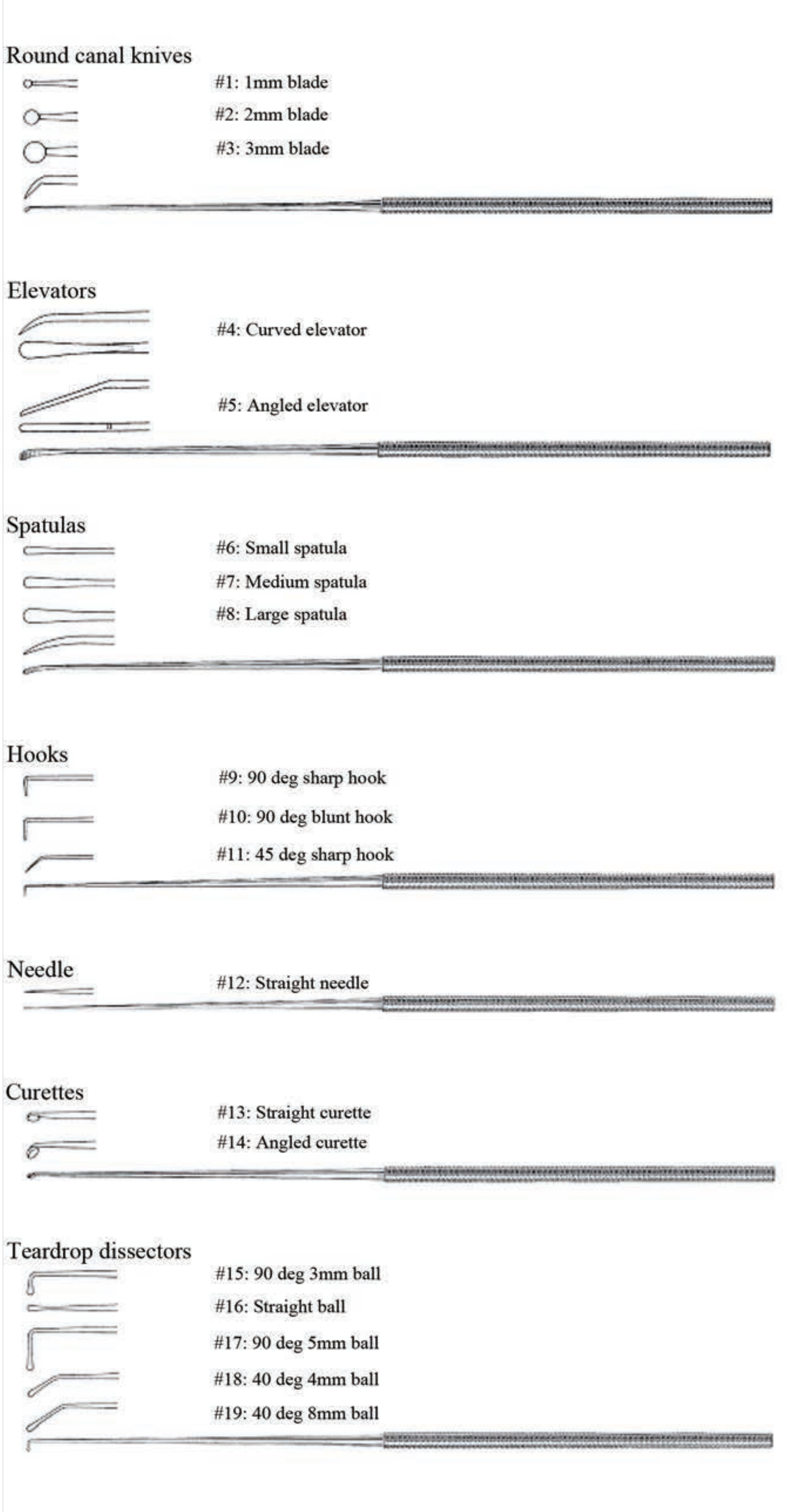

Round canal knives
#1: 1mm blade
#2: 2mm blade
#3: 3mm blade
Elevators
#4: Curved elevator
#5: Angled elevator
Spatulas
#6: Small spatula
#7: Medium spatula
#8: Large spatula
Hooks
#9: 90 deg sharp hook
#10: 90 deg blunt hook
#11: 45 deg sharp hook
Needle
#12: Straight needle
Curettes
#13: Straight curette
#14: Angled curette
Teardrop dissectors
#15: 90 deg 3mm ball
#16: Straight ball
#17: 90 deg 5mm ball
#18: 40 deg 4mm ball
#19: 40 deg 8mm ball

Fig. 1.3 Rhoton microscopic dissector set consists of round canal knives, spatulas, curettes, needle dissectors, and teardrop or ball-tipped dissectors in various sizes and angulations.

guidelines for brain clip biocompatibility and closing force measurement, developed after the only reported magnetic resonance imaging (MRI)-related fatality from an implanted vascular clip.[17] Concern regarding ferromagnetism induced by the metal-working process around steel clips has pushed the market for aneurysm clips such that essentially all currently used clips are made of cobalt- or titanium-based alloys.[18] While being MRI-safe, these clips also produce minimal susceptibility artifact. Ceramic clips have also been proposed for these advantages.

All modern clip appliers are crossed-leg levers. The bayonetted design is critical for deep and narrow corridors, and the tips may be straight, up-angled, down-angled, side-angled, or adjustable. Closure of the applier tips compresses the clip base and forces its blades to open. A locking mechanism may be present, similar to the Castroviejo needle holder, though many surgeons prefer disabling this feature to avoid accidental jamming and to preserve the smooth flow of the clipping maneuver.

The most popular aneurysm clips (the Sugita and Yasargil designs) function as crossed-action double levers.[19] Therefore, the closing force is not equally distributed throughout the length of the blade, but rather increases linearly from distal to proximal, so these clips are weakest at their tips. Additionally, the very nature of their crossed-action design results in the blades being slightly more separated distally than they are proximally even after full closure on the aneurysm neck. This discrepancy is accentuated by the thickness of the aneurysm wall. Several strategies are employed to counteract this, such as tandem clipping using a fenestrated clip, use of a reinforcing clip, or clip reconstruction using multiple clips.

Aneurysm clips are either temporary or permanent and miniature or standard in size. Some manufacturers color-code these categories for ease of recognition, typically using a gold color to indicate temporary clips. The closing force of a temporary clip is high enough to close the vascular lumen without crushing the cellular layers of the vessel. Placement of temporary clips must be well-planned so as to avoid obstructing the view of the aneurysm. A common scenario is to use a bayonetted clip for the arterial segment in the "near field," and a straight clip for the "far field" segment, and to apply clips from deep to superficial, to maintain visibility.

Permanent clips exert greater closing forces and often include interlocking serrations on the surface of the blades to prevent slippage on the vessel wall. Proper aneurysm clipping is usually a process of analyzing the configuration of the neck and related vasculature before it is clipped, and how it will change after it is clipped. Once flattened, a previously circular aneurysm neck will now have a length of approximately 1.5 times its original diameter. Blade length therefore needs to be approximately 50% longer than the unclipped neck. A vast array of clipping strategies has been developed to address challenging scenarios; however, the simplest appropriate clipping strategy is the goal in every case.

Another type of clip design reverses the traditional applier–clip interface.[20] Here, the applier is double-scissored. Flexion of the fingers results in opening of the applier tips, which fit inside the open head the clip, which functions as a scissoring spring. This set includes mono-shafted appliers as well, and offers the lowest profile among available aneurysm clip systems.

Sundt and Nofzinger developed a Teflon-lined encircling clip-graft in 1967, for use on blister aneurysms or segmental vascular defects or torn vascular walls.[21] This technique can be lifesaving in certain situations, and the graft can be substituted by using a firm gauze strip or Gore-Tex cut to size, then wrapped around the vessel wall defect, held in place with the right measure of tension by an aneurysm clip.

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2 How to Repair the Intracranial Aneurysm: Clipping or Coiling Decision Making

Menno R. Germans, Luca Regli, and R. Loch Macdonald

Abstract
When a patient presents with an intracranial aneurysm, there are at least four options for management: no follow-up, neuroimaging follow-up, neurosurgical clipping, or endovascular coiling. The goal of management should be to recommend and carry out the option that has the highest probability of giving the patient the longest, healthiest life possible. The decisions rest on estimates of the natural history of the aneurysm if left untreated or observed until it grows or ruptures or reruptures, the life expectancy of the patient, the risks and adverse effects of the management strategy such as treatment complications, need for neuroimaging follow-up if any, effects on quality of life if the aneurysm is not repaired, and the efficacy of the repair procedure. Myriad factors and sometimes even fragments of scientific data contribute to each of these overall estimates. Some cases are straightforward and clipping or coiling or either one can be highly recommended, whereas in other cases the factors and data conspire to challenge even the most experienced neurovascular/endovascular specialist.

Keywords: intracranial aneurysm, neurosurgical clipping, endovascular coiling, aneurysm repair

2.1 General Principles

Although aneurysm clipping generally provides the most reliable treatment with the best occlusion rates and lowest risk for postoperative aneurysm rupture, the morbidity is generally higher than endovascular coiling.[1,2] For unruptured aneurysms, there are no randomized clinical trials to guide decision making, and recommendations are based on natural history studies and expert opinion.[3] For ruptured aneurysms, there are at least four randomized clinical trials. In general, overall outcome is better with endovascular treatment of ruptured aneurysms that fit the features of those included in the randomized trials: small anterior circulation aneurysms in good-grade patients. On the contrary, certain aneurysms are treated better by one of the modalities, such as clipping of unruptured middle cerebral artery (MCA) aneurysms, although opinion is divided and is evolving even in the absence of scientific data.[4] Similarly, posterior circulation aneurysms, especially those of the basilar artery and its bifurcation, are mostly treated endovascularly. Overall, however, because of many factors that influence outcome of aneurysm treatment, there remains vigorous debate about the optimal treatment modality in individual patients. The decision for management modality of a specific aneurysm is best decided by a multidisciplinary team, including neurovascular surgeons and neurointerventional radiologists. Some of the controversies about the optimal treatment modality (microsurgical treatment vs. endovascular treatment) for a specific aneurysm/patient are described below.

2.1.1 Unruptured Intracranial Aneurysms

About 3% of the general population have an intracranial aneurysm, and most aneurysms never rupture.[5] Natural history studies show the risk of rupture increases with increasing aneurysm size, older patient age, history of hypertension, history of subarachnoid hemorrhage, and posterior circulation and anterior communicating artery (AcomA) location. Smoking is also probably important and hence, regardless of treatment of the aneurysm, patients should be advised to stop smoking and to optimize management of other cardiovascular disease risk factors. Symptomatic aneurysms were generally not included because they are almost always repaired due to the very high risk of rupture. These risks have been estimated from patients selected for no aneurysm treatment and then followed over time.[6,7,8] These data can be used to give a rough estimate risks of observation of the aneurysm. Risks of treatment are less well documented. Etminan et al developed an unruptured intracranial aneurysm treatment scale that assessed overall risks of treatment versus observation.[9] In general, the risks of treatment for clipping or coiling are even less well known. The decision to repair or not is based largely on the age and life expectancy of the patient, the anticipated risk of rupture, risk of aneurysm repair, expertise of the treating physicians, and desires of the patient.

2.1.2 Ruptured Intracranial Aneurysms

Systematic review and meta-analysis of four randomized trials of clipping versus coiling found that coiling was associated with better 1-year clinical outcomes.[2] Mortality was not significantly different. The International Subarachnoid Aneurysm Trial (ISAT) contributed almost 80% of the patients to this analysis.[10] The short follow-up and the numerous changes that have occurred in endovascular treatments, microneurosurgery, and neurointensive care have raised questions about these results. In addition, coiling is used to repair aneurysms that were not well represented in ISAT. Despite the fact that an increasing percentage of ruptured aneurysms are being repaired by coiling, these factors should be considered when making treatment decisions.[11]

2.2 Middle Cerebral Artery Aneurysms

The MCA remains the vessel with the most discussion between aneurysm treatment modalities. Much literature is written that supports either treatment, with a recent review summarizing the overall results.[4] One situation where surgery is most often superior to endovascular treatment is in the presence of a large or giant MCA aneurysm. The incorporation of major branches into the aneurysm sack makes the endovascular treatment a high-risk procedure with a high rate of incomplete occlusion, where on the contrary different surgical options exist with acceptable results.[12]

2.2.1 Ruptured MCA Aneurysms

According to a recent review, ruptured MCA aneurysms are best treated by coiling, which has the lowest rate of unfavorable outcome, although no firm conclusions could be drawn due to the variation in study design and lack of standardized reporting on MCA aneurysm treatments.[4] Nevertheless, certain situations make surgical treatment of this aneurysm the modality of choice, such as the presence of a broad-based aneurysm, or a significant subdural, intraparenchymal, or sylvian fissure hematoma.

When there is a large, space-occupying intracranial hematoma, the craniotomy needs to be large enough to expose the complete hematoma and allow for immediate decompression of the brain. In cases with a subdural hematoma, it can carefully be removed before proceeding with the aneurysm clipping procedure. Care must be taken to avoid excessive retraction of the arachnoid over the sylvian fissure and adjacent brain since the presence of subdural blood may mean the aneurysm has eroded through the arachnoid and is attached to the dura. When there is an intraparenchymal or sylvian fissure hematoma, we first make a large decompressive craniotomy with flattening of the sphenoid ridge so that the carotid artery can be identified with minimal brain retraction. After opening of the basal cisterns to release cerebrospinal fluid, the carotid artery is freed from arachnoid adhesions so that a temporary clip can be placed, if necessary. Next, we perform a small corticotomy at a noneloquent site and as close to the hematoma as possible, or we open the sylvian fissure superficially, to approach the hematoma. The hematoma is partially removed where some hematoma is left behind around the aneurysm to prevent a rebleed. After aneurysm clipping, we remove the remaining hematoma, keeping in mind that a hematoma in the sylvian fissure (identified as a serrated edge on the CT scan, indicating the insular gyri) might be difficult to remove (▶ Fig. 2.1a,b).

2.2.2 Unruptured MCA Aneurysms

In contrast to ruptured MCA aneurysms, unruptured aneurysms are better treated surgically, with less complications, better occlusion rates, and less retreatment rates.[4] Some morphological aspects on the preoperative angiography can facilitate the surgical strategy, where a shorter M1 segment, larger M1 angle opposed to the skull base, and posteroinferior aneurysm projection are related to a higher risk for surgical complications (**Video 2.1**).

2.3 Posterior Communicating Artery Aneurysms

When a posterior communicating artery (PcomA) aneurysm can be treated by both surgery and coiling, the latter modality tends to result in better functional outcome. Nevertheless, these aneurysms often have a wide neck and/or the PcomA originates at the neck of the aneurysm, making an isolated coiling procedure more risky. An alternative is the stent-assisted or balloon-assisted technique, with higher complication and lower obliteration rates, especially in PcomA aneurysms.[13,14] Thanks to the technical advances in neurosurgery, including improved microsurgical techniques and approaches by minicraniotomies, clipping a PcomA aneurysm has become a treatment with low risks and acceptable morbidity. It might therefore be a good alternative when a stent or balloon is necessary to coil the aneurysm, especially in case of ruptured aneurysms.

2.3.1 Posterior Communicating Artery Aneurysm with Oculomotor Deficits

When a PcomA aneurysm projects inferiorly, it can cause a (partial) oculomotor deficit (**Video 2.2**). Both treatment modalities can improve the deficits by either removing the compression on the nerve by surgical clipping or reducing the pulsations on the nerve by coiling. A review comparing both modalities in respect to oculomotor nerve palsies due to PcomA aneurysms shows that it resolves in a significant higher proportion of patients after surgical clipping.[15]

In general, we prefer not to open unruptured aneurysms after they are clipped, but when clipping a PcomA aneurysm with oculomotor compression, we tend to puncture the aneurysm after occlusion, shrink it by bipolar coagulation, or cut out the dome of the aneurysm.

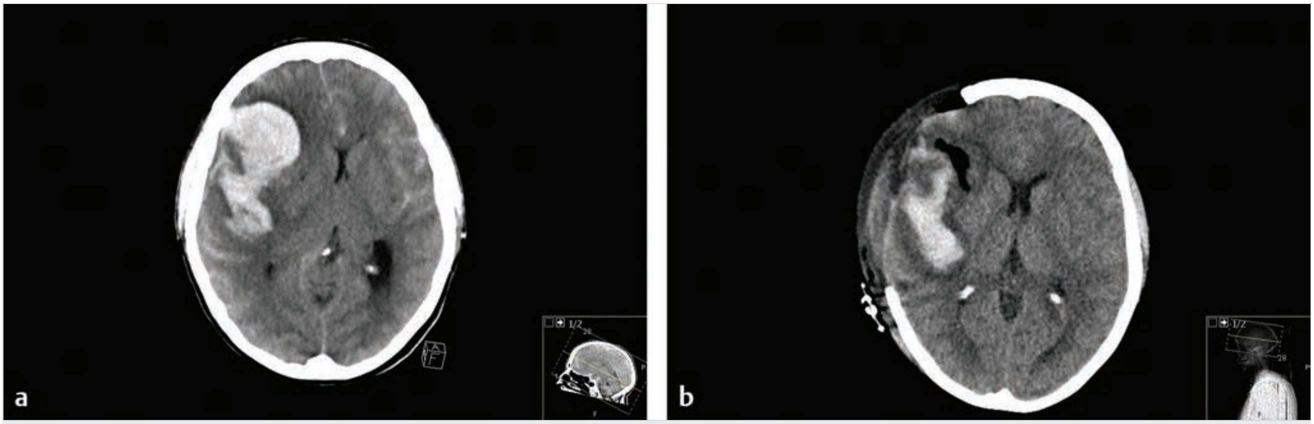

Fig. 2.1 **(a)** Preoperative CT scan with intraparenchymal and sylvian hematoma due to ruptured right middle cerebral artery aneurysm. **(b)** Postoperative CT scan after decompressive craniectomy, middle cerebral aneurysm clipping, and evacuation of intraparenchymal hematoma. The sylvian hematoma was not removed due to its tenacity and risk for damaging sylvian fissure arteries.

2.4 Anterior Communicating Artery Aneurysms

Clipping an AcomA aneurysm is challenging due to the difficult exposure of the vascular anatomy, complex angioarchitecture, and flow dynamics (**Video 2.3**).[16] An endovascular procedure might therefore be a good alternative with proven good functional outcome.[17] Sometimes the endovascular procedure is technically not possible or a higher risk for complications can be expected, so that microsurgical clipping still is a good alternative. Consequently, the most difficult AcomA aneurysms are treated surgically in our practice, making the procedure even more demanding with a higher risk for complications. We therefore try to treat AcomA aneurysms endovascularly and perform surgery only in selected cases.

2.5 Pericallosal Artery Aneurysms

Distal anterior cerebral artery aneurysms, which are most commonly pericallosal artery aneurysms, are often small and broad-based with a branch originating at the base of the aneurysm (**Video 2.4**). This, together with the distal location, small parent vessel diameter, and high recurrence rate, makes them challenging to treat endovascularly.[18] The most common surgical approach is through the interhemispheric fissure via a right-sided parasagittal craniotomy, tailored to the location of the aneurysm in relation to the genu of the corpus callosum.[19] Because the surgical treatment is associated with low complication rates, we prefer to clip a pericallosal artery aneurysm when the endovascular approach is expected to be challenging, or when a hematoma with significant mass effect is present (in about 25% of cases).

2.6 Posterior Inferior Cerebellar Artery Aneurysms

An aneurysm on the posterior inferior cerebellar artery (PICA) is usually located at the origin of the artery from the vertebral artery but can also originate distally, where it tends to be more often fusiform with a higher risk for a rebleeding.[20] The location of the aneurysm in the vicinity of lower cranial nerves makes a clipping procedure challenging with high rates of postoperative lower cranial nerve deficits.[21,22] Other surgical techniques, such as PICA-to-PICA or occipital artery-to-PICA bypass with occlusion of the parent artery, exist, but these techniques are complex and require an experienced vascular surgeon (**Video 2.5**). The endovascular treatment options include (stent or balloon-assisted) coiling of the aneurysm or parent vessel occlusion, which is mostly done with dissecting aneurysms and often tolerated without severe neurological deficits. Although endovascular treatment seems to cause less complications than surgery, the incomplete occlusions and retreatment rates are high.[21,22] This implies that both treatment modalities have their drawbacks and the optimal treatment modality should be decided by an experienced multidisciplinary team.

2.7 Carotid Blister-Like Aneurysms and Other Special Cases

A carotid “blood blister-like” aneurysm is not a true aneurysm, but consists of a combination of blood clots, fibrous tissue, and adventitia (**Video 2.6**). The rerupture and growth rates are high, as is the mortality. Different surgical strategies exist, such as direct clipping, wrapping, and parent artery occlusion with or without bypass.[23] All these surgical strategies are associated with significant morbidity and mortality, including a high intra- and postoperative rebleed rate. Endovascular treatment of carotid blister-like aneurysms, by means of coils with or without stenting, also is associated with substantial morbidity and mortality with a high rate of rebleeds and incomplete occlusions. More recently, flow-diverting stents are used in these cases, possibly with better results, indicating that this may be a promising strategy.[23] Nevertheless, if flow-diverting stent treatment fails, surgery might still be a feasible option.

2.8 Ophthalmic Segment Aneurysms

Aneurysms of the ophthalmic segment of the internal carotid artery are risky to treat because of the arising ophthalmic artery and proximity to the optic apparatus. Surgical treatment options include direct aneurysm clipping, where anterior clinoidectomy, dissection of the aneurysm, and mobilization of the optic nerve can induce or worsen visual deficits and can cause additional complications.[24] On the other hand, embolization of ophthalmic segment aneurysms, with direct coiling or flow-diverting stents, has lower occlusion rates and can cause ophthalmic artery thrombosis and retinal emboli with deterioration of visual function in 18 to 26%.[25] In summary, visual outcome is probably better with microsurgery and occlusion rates may be higher. Nevertheless, the approach to the ophthalmic segment aneurysm is challenging and general complications do occur more often than in endovascular treatments.

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3 Aneurysm Surgery Techniques

Gregory J. Zipfel and Ralph G. Dacey, Jr

Abstract

The goals of intracranial aneurysm surgery are to obliterate the aneurysm while preserving flow through the parent vessel and associated perforators, minimize manipulation and retraction of the brain and avoid postoperative complications. This chapter reviews the surgical instrumentation, neuroanesthetic principles, and general intradural surgical techniques required for achieving optimal results with direct surgical aneurysm obliteration.

Keywords: aneurysm, clipping, technique, intracranial aneurysm, microscope, microsurgery, neuroanesthesia

3.1 Operative Equipment

The cornerstone of any operation for an intracranial aneurysm is the operative microscope. The microscope should be properly balanced and controlled with a foot pedal or mouthpiece. It is helpful to project the operative image through an intraoperative video monitor. To facilitate microsurgical movement, the surgeon's hands need to rest on a hand rest or the patient's head to remove the influence of large proximal motor units in the surgeon's arms. The patient's head must be firmly fixed in the appropriate operative position using a pin head holder attached to the surgical table. A radiolucent head holder aids intraoperative cerebral angiography. A self-retaining retractor system with flexible arms and tapered retractor blades is used although the use of fixed retractor blades should be minimized. Microsurgical instruments needed include bipolar, usually irrigating, forceps of varying lengths and tip widths, suction tips of varying sizes, preferably with thumbholes that permit surgeon control of suction strength, straight and curved microscissors, and dissecting spatulas and hooks in several shapes and sizes.

A comprehensive set of aneurysm clips and clip appliers is critical, including several of each clip type for techniques such as tandem clipping with identical aneurysm clips. Clip appliers also come in a variety of configurations, which is important in allowing maximum degrees of flexibility to the aneurysm surgeon during clip application.

Because direct inspection does not always accurately confirm that the surgery has been successful, many surgeons use adjuncts to demonstrate complete aneurysm occlusion and patency of parent and branch arteries. These include intraoperative endoscopy, Doppler ultrasonography, intraoperative angiography, and near-infrared indocyanine green videoangiography. Aneurysm surgeons should familiarize themselves with all of these adjuncts and find which ones they prefer. Frame and frameless stereotactic techniques are not required for most intracranial aneurysms; however, they may be useful for distal anterior cerebral artery aneurysms.

3.2 Neuroanesthesia, Monitoring, and Brain Relaxation

The keys are to avoid marked blood pressure fluctuations throughout the operation, avoid maneuvers that lower cerebral perfusion pressure or increase cerebral metabolism and thus increase the risk of cerebral ischemia, and maximize brain relaxation. Osmotic diuresis and mild hyperventilation are often used to achieve the latter. The anesthesiologist should always be prepared for massive blood loss. For proximal carotid aneurysms where intracranial proximal control may not be achieved early on, they should know where the carotid artery is in the neck for compression if necessary.

During temporary clipping, additional maneuvers are sometimes employed for cerebral protection. Propofol, pentobarbital, or etomidate may be administered to achieve burst suppression on electroencephalography (EEG). The arterial pressure is also often increased. Intraoperative moderate hypothermia was not neuroprotective in one trial of good-grade patients with subarachnoid hemorrhage. The rest of these techniques have not been studied in randomized clinical trials.

3.3 Operative Procedure for Intracranial Aneurysms

Once the bony exposure is complete and the dura is opened, the surgeon should assess the adequacy of brain relaxation. If insufficient, additional relaxation can often be achieved through a ventriculostomy or lumbar drain. Once adequate brain relaxation has been achieved, the operative microscope is brought into the field.

Microsurgical dissection of the aneurysm starts with obtaining proximal and distal arterial control followed by preparation of the aneurysm neck for clipping. Dissection usually involves retraction with suction and cottonoids, spreading the arachnoid with bipolar forceps, and sharp arachnoid dissection. Arachnoid that is not easily separated has to be cut with a knife or microscissors. Typically, the suction tip can be used to create gentle traction upon the arachnoid bands, which can then be divided sharply with microscissors or an arachnoid knife. The suction should be regulated to be able to suction fluid without injuring the pia.

It is important to attain proximal arterial control prior to dissection and manipulation of the aneurysm. For many paraclinoid aneurysms and the occasional posterior communicating artery aneurysm that arises from a foreshortened carotid, it may be difficult to achieve proximal control, and it may need to be obtained by isolation of the cervical carotid artery. Distal control is the next step and involves the same techniques as for establishing proximal control. Following dissection of the proximal and distal vessels, including preparation for temporary clipping, the surgeon dissects the arachnoid planes around the fundus and neck of the aneurysm. Retraction of the aneurysm with the blunt suction tip will place arachnoidal bands on stretch, permitting sharp division with microscissors or an arachnoid knife. Blunt tearing of arachnoid bands should be avoided to minimize risk of intraoperative aneurysm rupture. It is generally advisable to begin separation of surrounding branches and the wall of the parent vessel from the midportion of the fundus, carefully developing a plane and extending the dissection proximally from the fundus to the aneurysm neck. Dissection more distally toward the aneurysm dome, on the other hand, should be avoided. Once a plane is developed, a smooth dissector is passed on either side of the aneurysm neck to simulate passage of an aneurysm clip blade. No force should be needed, and the aneurysm should not move.

3.3.1 Temporary Arterial Occlusion

Some surgeons use temporary occlusion in all cases. Most use it selectively for large or complex aneurysms and for intraoperative aneurysm rupture. This is almost always done with temporary clips. Adjuncts to protect the brain during temporary clipping may be employed as already discussed. The principles are to minimize the time of temporary clipping, apply the clips in a manner that avoids obscuration of the aneurysm, and avoid occluding perforating arteries. Proximal occlusion only is usually adequate, but trapping may be needed to control aneurysm rupture or for large and giant aneurysms that need to be opened or collapsed for clipping.

For proximal carotid aneurysms, a suction decompression technique can be used (▶ Fig. 3.1). The aneurysm can also be decompressed by inserting a needle into it when it is trapped and then sucking it out (▶ Fig. 3.1). Keep in mind that, like opening the aneurysm, this creates a point of no return where the aneurysm has to be occluded, which may be fine for ruptured lesions but bears consideration when operating on asymptomatic, unruptured lesions.

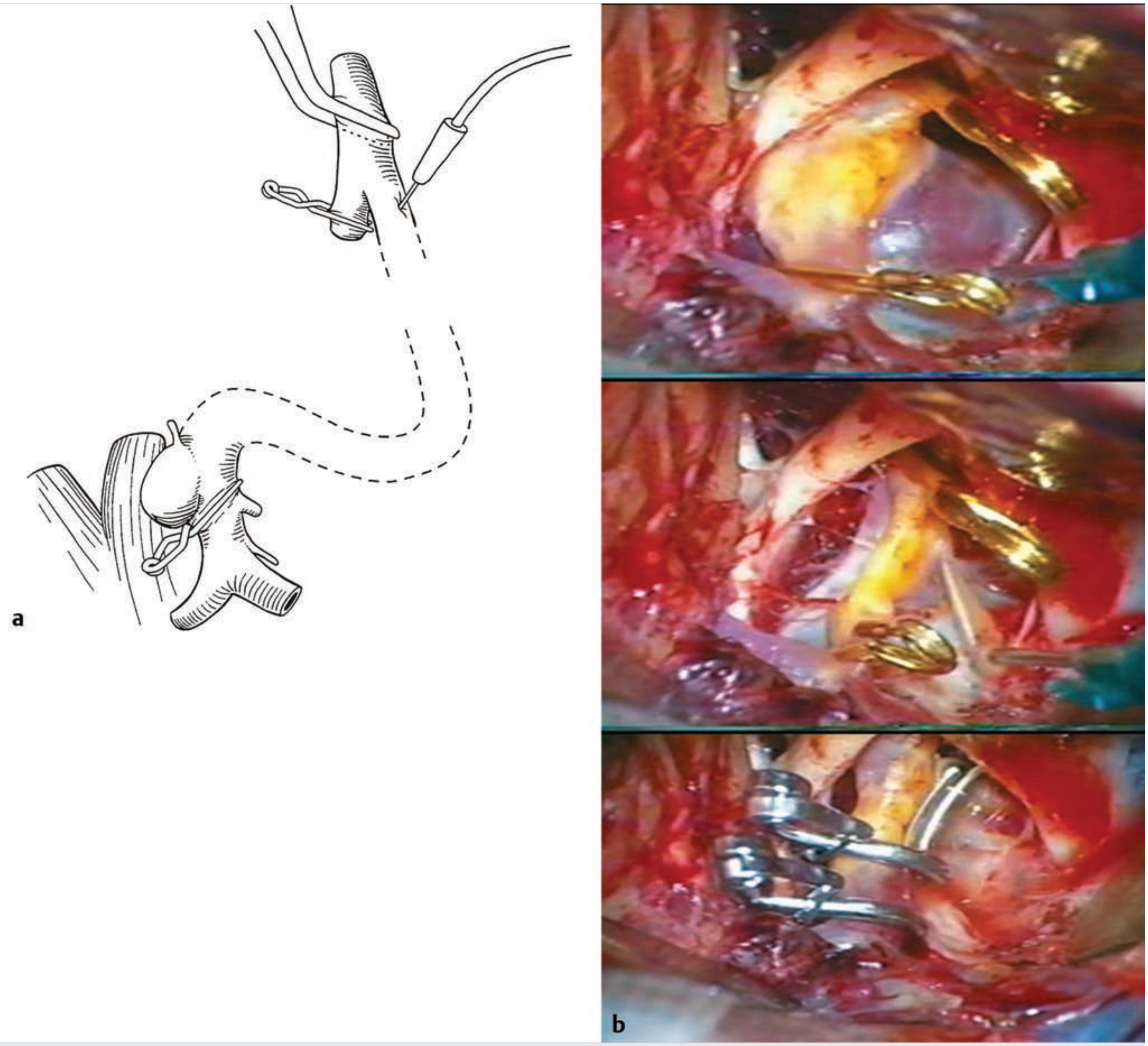

Fig. 3.1 Suction-decompression technique. **(a)** “Dallas” technique for proximal internal carotid aneurysms. The extracranial cervical carotid and intracranial carotid arteries distal to the aneurysm are temporarily clipped and the aneurysm is aspirated through a catheter inserted into the extracranial carotid. **(b)** Needle decompression technique. Similar suction decompression can be achieved by first trapping the intracranial arterial segment and then puncturing the aneurysm dome with a “butterfly” needle connected to suction (▶ Fig. 3.1a reproduced with permission from Roberts GA, Dacey RG Jr. General techniques of aneurysm surgery. In: Le Roux PD, Winn RH, Newell DW, eds. Management of Cerebral Aneurysms. Philadelphia, PA: Saunders; 2004:563–582.)

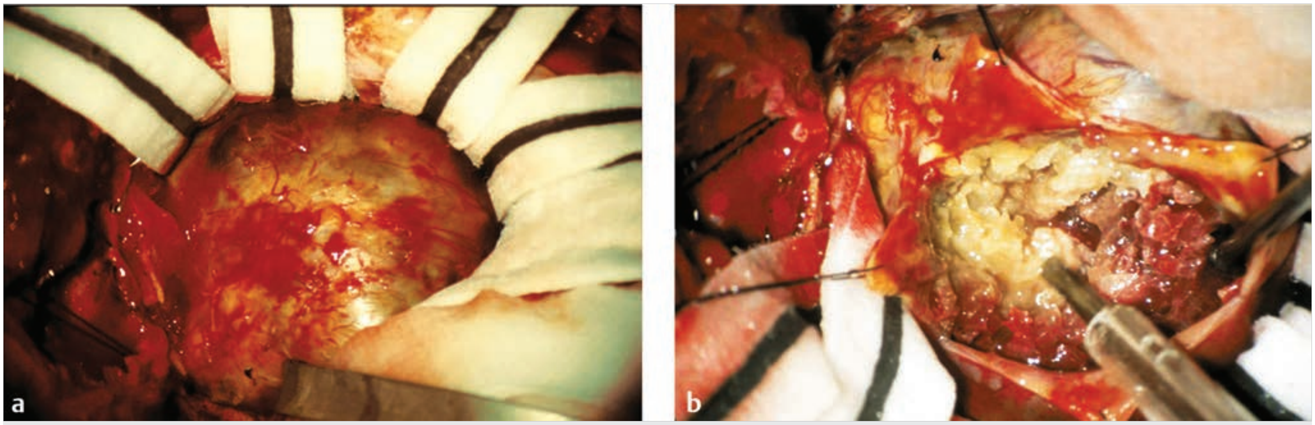

Fig. 3.2 Intraluminal thrombus. **(a)** Giant middle cerebral artery aneurysm with intraluminal thrombus. **(b)** After trapping of the arterial segment, the aneurysm dome is opened in cruciate fashion and intraluminal thrombus is removed with an ultrasonic aspirator.

3.3.2 Intraoperative Aneurysm Rupture

This is better to avoid than to deal with. It can occur during initial aneurysm exposure (predissection), during dissection of the aneurysm itself (dissection), or during aneurysm clipping (clipping). Rupture during the predissection phase is often a catastrophic event requiring aggressive measures for obtaining proximal arterial control. Brain swelling may mandate resection of the brain to gain access. Prevention is by maintaining adequate anesthesia and stable blood pressure and avoiding excess retraction during initial aneurysm exposure.

Aneurysm rupture during aneurysm dissection usually results from blunt dissection techniques or removal of hematoma around the aneurysm fundus or dome. It sometimes occurs while using optimal technique. Minor bleeding can be tamponaded with a small cottonoid and suction. Heavier bleeding generally requires temporary clipping. The authors use two suckers to clear the field of blood, permitting the surgeon to identify the site of rupture, and then place a single sucker immediately on the rent to prevent or minimize blood escaping into the subarachnoid space obscuring the vascular anatomy. Controlled maneuvers are necessary so as not to tear the aneurysm further. Aneurysm rupture during clip application is usually managed by completing the clipping. Problems arise when the clip blades are not completely across the aneurysm and a tear occurs near the tips of the blades. This is prevented by making sure the neck is fully dissected and arachnoid bands are cut before clipping.

3.3.3 Management of Thrombus and Calcification

Intra-aneurysmal thrombus can be insignificant as in the plug of clot in the dome of a small aneurysm, or constitute an enormous obstacle to aneurysm clipping. It is a factor in the decision to clip or coil because abundant thrombus makes recanalization after coiling more likely. Preoperative computed tomography (CT) and magnetic resonance imaging (MRI) will define any substantial thrombus and should be done if it is suspected. If there is a lot of thrombus, it may be necessary to open the aneurysm and remove it to allow clipping (▶ Fig. 3.2). The aneurysm usually has to be trapped and cut open. An ultrasonic aspirator may aid removal of firm clot.

The incidence of calcification and significant atheroma within cerebral aneurysms rises significantly with lesion size. Preoperative fine-cut CT scans through the aneurysm will often identify calcified atheroma. Calcification increases the difficulty and risk of surgery and is a factor in deciding whether surgical or endovascular approach is more appropriate. An intraoperative consideration in these cases is that one may need to place the aneurysm clip more distally along the aneurysm neck so as to minimize the chance of parent vessel stenosis or occlusion (▶ Fig. 3.3). A single aneurysm clip may not have enough force to close the neck or it may close the thick part of the wall but blood may still enter the aneurysm through a patent lumen between thin parts of the wall. Options here are the fenestrated clip, booster clip, and stacking multiple clip techniques. The clip may slide down and occlude the parent artery. Options here are to apply clips more distally and then remove the first clips.

3.3.4 Strategies for Definitive Clip Application

The aneurysm clip blades should usually be placed parallel to the long axis of the neck of the aneurysm along the parent vessel wall. This minimizes the risk for parent vessel encroachment and reduces the risk of an inadvertent shearing of the aneurysm at its base. However, some aneurysms, such as posterior communicating artery aneurysms, are routinely clipped with the blades perpendicular to the long axis of the parent carotid artery. Use the simplest straight clip possible before resorting to complicated strategies and estimate the correct length as about 25% longer than the neck of the aneurysm. Use temporary clipping when trouble arises.

Ventrally projecting internal carotid, basilar bifurcation, superiorly projecting anterior communicating, and large/giant thick-walled aneurysms may be best reconstructed with fenestrated clips (▶ Fig. 3.4). The shank clip technique can be useful when an aneurysm arises at the bifurcation of an artery and also along one or both sides of the bifurcation such that use of a straight aneurysm clip will obliterate only the main portion of

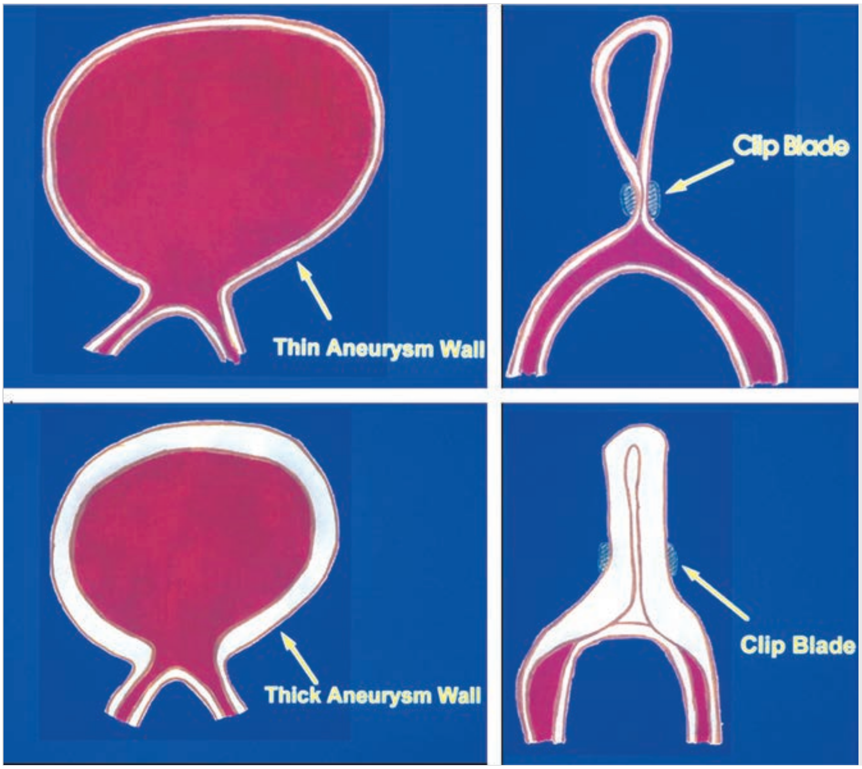

Fig. 3.3 Atheroma. Special clip strategies must be used when significant atheroma is encountered during aneurysm surgery because standard clipping techniques will often lead to parent vessel stenosis or occlusion related to the thickened atheromatous wall, as depicted.

the aneurysm while a significant remnant or “dog-ear” remains (▶ Fig. 3.5).

Large or giant aneurysms with broad necks or high transmural pressure and wall tension often require multiple clips applied in a variety of “tandem” clip configurations. The most straightforward is placing a duplicate clip just distal to the initial clip. Another method is to put a fenestrated straight clip in tandem with a nonfenestrated straight clip to focus additional closing forces directly on the distal aneurysm neck (▶ Fig. 3.6). Advancement clip strategies can also be useful. If there is premature rupture, a clip can be deliberately applied distal to the aneurysm neck to occlude the majority of the aneurysm sac, including the dome where aneurysm rupture is most likely (▶ Fig. 3.6). Aneurysm dissection is then completed and a proximal tandem clip placed across the aneurysm neck for definitive obliteration. In the opposite case, wherein initial clip placement produces parent vessel compromise, a second clip can be placed just distal to the first, allowing removal of the first clip.

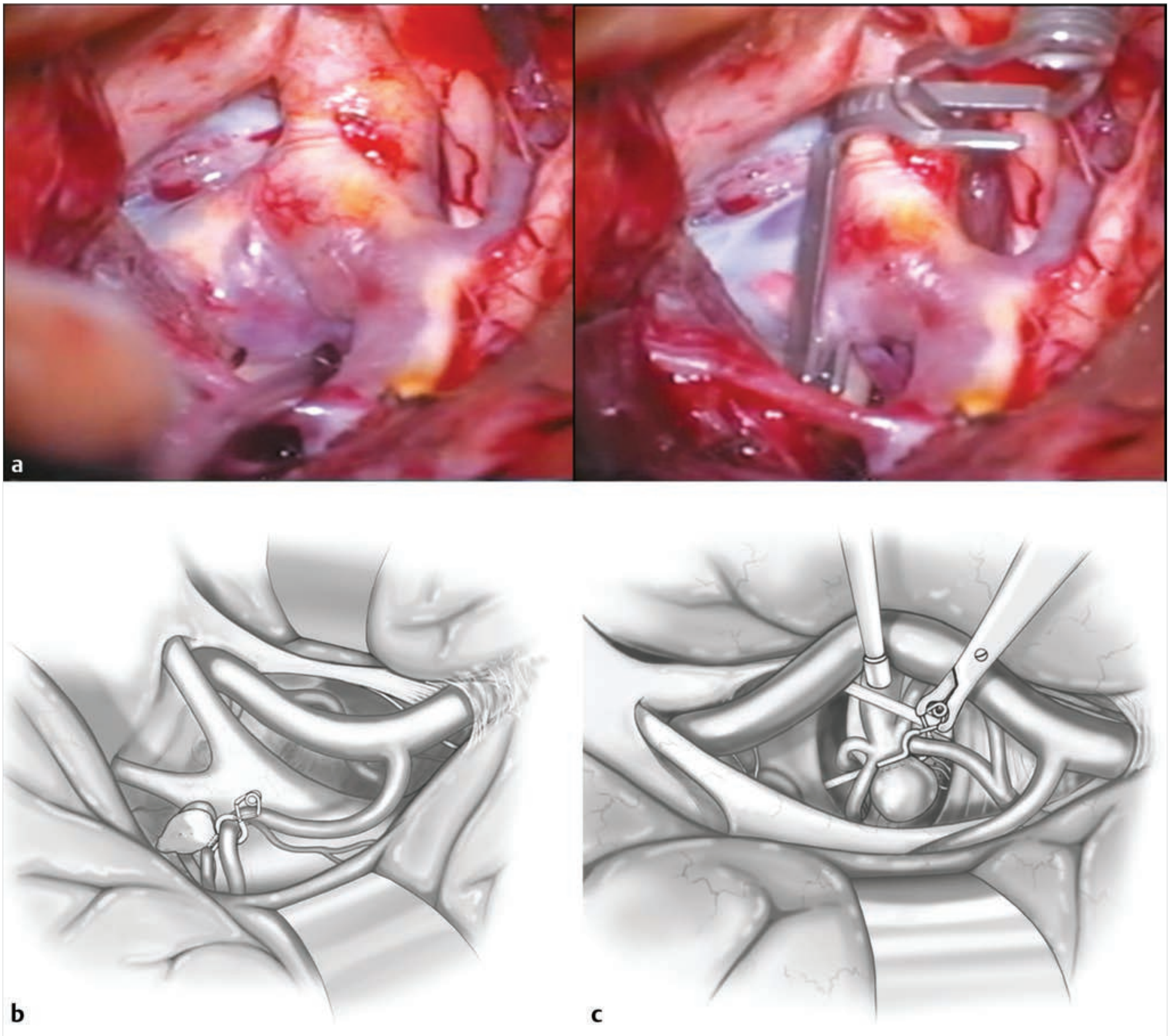

Fig. 3.4 Fenestrated clips permit encircling a parent or branch artery while obliterating the aneurysm neck. Three common uses are **(a)** a posterior carotid artery wall aneurysm, **(b)** a superiorly projecting anterior communicating artery aneurysm, and **(c)** a basilar apex aneurysm.

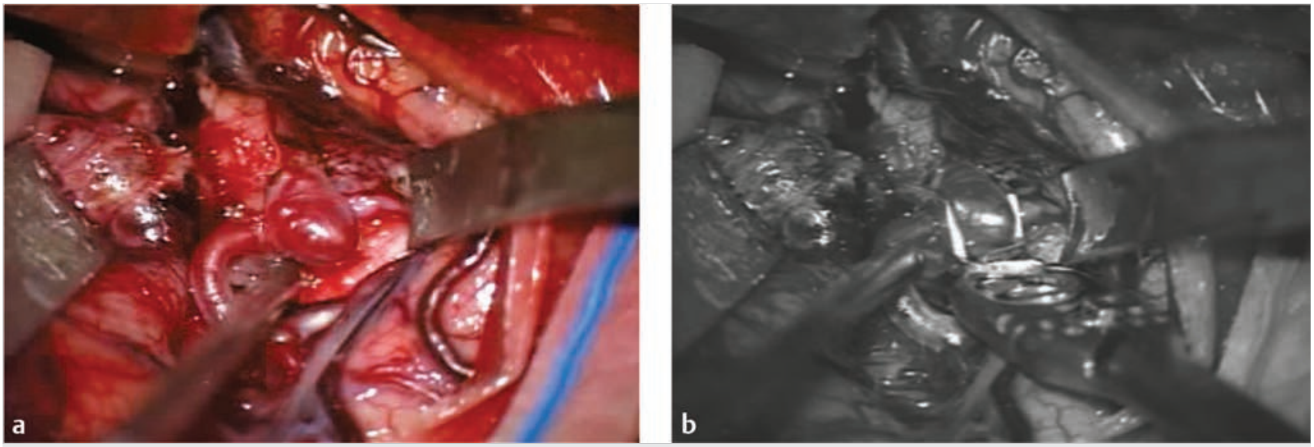

Fig. 3.5 **(a)** A middle cerebral artery bifurcation aneurysm. **(b)** A shank clip allows its apex to accommodate the main channel of the parent vessel, while its angled portions obliterate the aneurysm lobes that extend to the sides of the bifurcation.

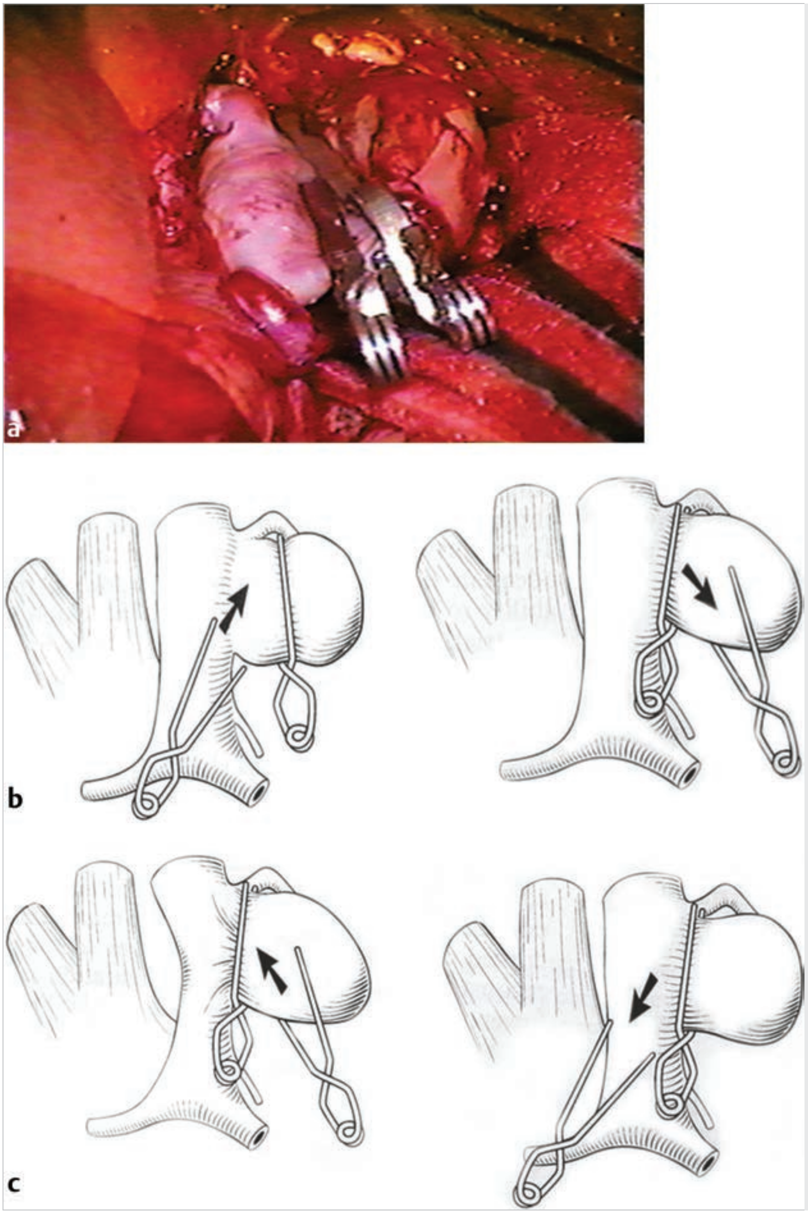

Fig. 3.6 Tandem and advancement clipping techniques. **(a)** Tandem clip technique used when a single clip does not have sufficient closing force to obliterate the aneurysm. A fenestrated clip in tandem allows additional closing force directly applied to the distal aneurysm neck. **(b)** Advancement clip techniques where clip advancement proceeding from distal to proximal along the aneurysm is used to control intraoperative rupture or when the neck is initially obscured. **(c)** Reverse clip advancement can be used when initial clip placement kinks the parent artery. (Reproduced with permission from Roberts GA, Dacey RG, Jr. General techniques of aneurysm surgery. In: Le Roux PD, Winn RH, Newell DW, eds. Management of Cerebral Aneurysms. Philadelphia. PA: Saunders; 2004:563–582.)

4 Pterional Approach

R. Loch Macdonald

Abstract

The pterional or frontotemporal craniotomy is a very common and widely used approach since it can be used for all of the common anterior circulation and upper basilar artery aneurysms as well as for other vascular and nonvascular indications. There are many variations in the basic technique, as well as additions including expanding the approach through orbital and zygomatic osteotomies, extension of the incision posteriorly in order to create a decompressive hemicraniectomy, and various methods to remove the anterior clinoid process. The craniotomy also can be reduced in size as described in many different keyhole approaches centered on the pterion. One method for conducting a pterional craniotomy is described here.

Keywords: aneurysm, cerebral aneurysm, craniotomy, frontotemporal, pterional

4.1 Indications and Alternatives

A frontotemporal or pterional craniotomy is the main route of approach for aneurysms located on the internal carotid artery, the precommunicating segment (A1) of the anterior cerebral artery up to and including the anterior communicating artery complex, and the sphenoidal (M1) segment of the middle cerebral artery. Proximal internal carotid artery aneurysms often require intra- or extradural removal of the anterior clinoid process. Exposure of the cervical carotid artery may be used to gain proximal control in these cases. The pterional approach also can be used for aneurysms of the upper basilar artery, the details of which depend mostly on the rostral caudal location of the aneurysm in relation to the posterior clinoid processes. The craniotomy can be expanded by orbital rim and zygomatic osteotomies. These maneuvers tend to be indicated when basilar artery bifurcation is high and to widen the field in order to allow more illumination and more room to manipulate instruments. The main alternative to a pterional route for upper basilar artery aneurysm is a subtemporal approach. An anterior interhemispheric approach can be used for anterior communicating artery aneurysms.

4.2 Anatomy

The pterion is the small area on the lateral surface of the skull where the frontal, parietal, greater wing of the sphenoid, and squamous part of the temporal bone meet.[1] At this point, on the inner surface of the skull is the fused portion of the greater and lesser wings of the sphenoid. This ridge separates the anterior and middle cranial fossae and a portion generally is removed extradurally. The greater wing of the sphenoid forms the anterior wall of the middle fossa, whereas the lesser wing forms the posterior part of the floor of the anterior fossa. It juts posteriorly into the stem of the sylvian fissure between the basal surface of the frontal lobe and anteromedial temporal lobe.

4.3 Preoperative Preparation

Preoperative preparation includes an initial surgical checklist. The patient cannot be anesthetized until factors including ensuring the correct patient and side of approach and presence of the necessary neuroimaging and equipment including operating microscope equipped with indocyanine green fluorescence and mobile fluoroscopy for intraoperative angiography are taken into account. The patient is anesthetized and administered the preoperative antibiotics. Particular attention should be paid during induction of anesthesia and from here forward to preventing undue fluctuations in blood pressure, which might increase the chances of aneurysm rupture, especially when conducting surgery for a ruptured aneurysm. The initial positioning is done, consisting of flexing the operating table and reverse Trendelenburg tilting so that the head is up about 15 degrees in order to optimize venous drainage and brain relaxation. The head is fixed in a radiolucent head holder to permit intraoperative angiography.

The head position in the standard position for a pterional craniotomy is rotated about 30 degrees, raised up above the body and extended so the orbital rim and malar eminence are at the same level horizontally (▶ Fig. 4.1). More rotation is sometimes used for a middle cerebral artery aneurysm and less for an anterior communicating artery aneurysm. A shoulder roll under the ipsilateral shoulder may be needed to facilitate turning of the head. The degree of extension described is optimal when performing surgery with the diploscope attachment on the operating microscope because the surgeon and assistant stand on opposite sides of the head. When using a single binocular attachment with a side extension, the head can be extended more if desired but too much extension will make it difficult to see as far up the anterior fossa base. Another consideration is preparing the anterior neck and the carotid bifurcation area for proximal control for proximal internal carotid artery aneurysms or for possible bypass.

An option is to register the patient to use intraoperative neuronavigation. This is not absolutely necessary. It does have some value for teaching and showing trainees how to position the head to get a good trajectory toward the aneurysm and to facilitate insertion of an external ventricular drain (EVD) catheter, which often is required for ruptured aneurysm cases. Also, the frontal sinus can be delineated, and if it is large and the craniotomy is likely to enter it, a pericranial flap can be dissected from the skull from the superior temporal line up to the medial part of the incision.

It is adequate to only shave a line of hair along the incision (▶ Fig. 4.1). I often do an angiogram by retrograde injection through the superficial temporal artery so this can be palpated to locate it prior to marking the location of the incision. If the aneurysm is ruptured and insertion of an EVD is anticipated, then shave an additional swath of hair back from the incision at the level of the superior temporal line. An EVD may already have been inserted, although an advantage of waiting until craniotomy, if the patient's neurological condition permits, is that there may be more cerebrospinal fluid to drain leading to better

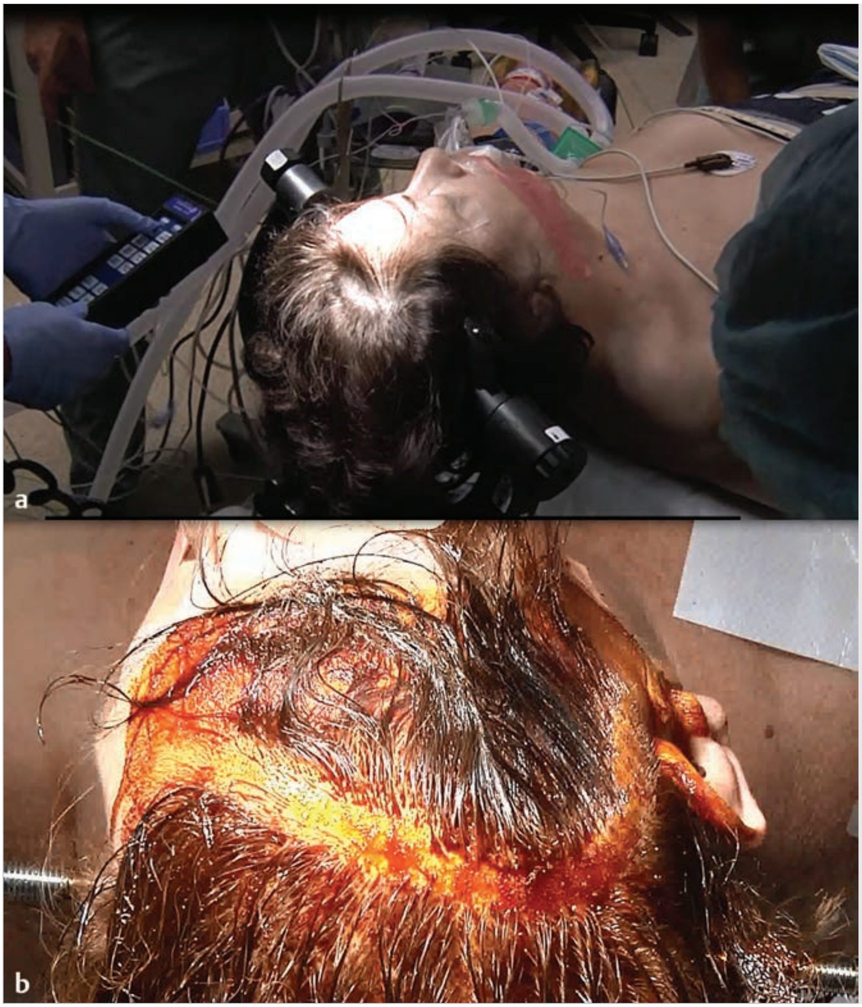

Fig. 4.1 **(a)** Positioning for a pterional craniotomy, with the operating table flexed so that the head is elevated 15 degrees and is above the heart. **(b)** The head is slightly extended and rotated about 20 degrees and only a thin line of hair shaved along the planned incision.

brain relaxation. The skin and hair are prepared with povidone iodine solution. This must be in contact with the skin for 3 or more minutes so it has time to dry.

For unruptured cases, I usually infiltrate the skin and subcutaneous tissue with about 8 mL of 1% xylocaine without epinephrine. This is avoided in patients with ruptured aneurysms because of the risk of affecting the blood pressure by an inadvertent intravascular injection. Nimodipine is commenced after diagnosis in patients with subarachnoid hemorrhage (SAH). The usual oral dose is 60 mg every 4 hours. Maintenance of normovolemia is imperative in the perioperative phase. There is no uniform agreement on choice of fluid replacement, but our practice is to use 0.9% normal saline with close monitoring of electrolytes and sodium and potassium supplementation as necessary. Anticonvulsants such as phenytoin may be administered to patients who have had a seizure, who are at high risk of having one (intracerebral hematoma), or who would be potentially harmed by one (poor-grade patients, those with already increased intracranial pressure). There is no evidence to support giving anticonvulsants to all craniotomy patients.

Smooth induction of anesthesia without altering the blood pressure is important so as to reduce the risk of aneurysm rupture or rerupture. The head pins should only be applied when it can be assured that the patient is adequately anesthetized. We do not use lumbar drainage but place an EVD preoperatively in patients with neurological symptoms and signs due to hydrocephalus or intraoperatively through the craniotomy when additional brain relaxation is needed.[2] An arterial line and urinary catheter are used in all patients. Central venous lines are optional. Always be prepared for massive uncontrolled bleeding. Intraoperative hypothermia was not beneficial in a large, randomized study of good-grade patients with SAH, so despite experimental data to the contrary, its use has to be considered questionable at this time.

For complex or giant aneurysms where prolonged temporary clipping or bypass procedures may be necessary, we use electroencephalography monitoring so we can induce burst suppression if necessary.

Also, we insert a ventricular drain into the frontal horn of the lateral ventricle once the dura is open in almost all patients with SAH. This is done by aiming perpendicularly to the brain from the top of a triangle 2.5 cm back along the sylvian fissure and 2.5 cm superiorly.[2]

4.4 Operative Procedure

4.4.1 Scalp and Temporalis Muscle

The incision starts temporally 0.5 to 1.0 cm anterior to the tragus and not more than 1.0 cm below the zygomatic arch. It curves up to or across the midline at the hairline. An angiogram will usually be done through the superficial temporal artery, so care should be taken not to injure it during the initial incision. Once a short incision is made, isolate the superficial temporal artery using Metzenbaum scissors. Once a segment is freed, tie a 3–0 absorbable suture loosely around it to mark it so that it can be located once the scalp flap is reflected. The rest of the incision is completed in stages. It is optimal to avoid incising the temporalis muscle fascia initially. The pericranium above the superior temporal line can be incised so that it can be reflected with the scalp flap unless entrance into the frontal sinus is anticipated. The skin is incised with a scalpel and the deeper layers with a scalpel or electrocautery. Scalp clips, such as Raney clips, are placed starting at the end of the incision so they can be stacked up close together with no spaces in between. We only make short segments of incision before applying clips in order to minimize blood loss. If stray hairs get in the incision, do not pull them since this will pull in more and more hair. Simply cut them with scissors.

Once the scalp is open down to the pericranium, the temporalis muscle needs to be incised and reflected, a procedure for which there are numerous variations (▶ Fig. 4.2). The author prefers to reflect the scalp and muscle as one unit. The advantage is that this minimizes the risk of injuring the frontalis branch of the facial nerve. The disadvantage is there is more muscle anteriorly that may hinder visualization along the sphenoid ridge. Initially, incise the temporalis fascia and pericranium, and start reflecting it with a flair or similar instrument. If one starts temporally and sweeps up, it is easier to preserve the deep layer of fascia under the muscle. It is also helpful to leave a cuff of muscle along the superior temporal line to sew the muscle back up to. The scalp and temporalis muscle are reflected forward as one layer. There are many other options including an interfascial approach and addition of bone removal of the orbital rim and zygoma. If an interfascial approach is chosen, the scalp is reflected before incising the temporalis fascia. As the scalp dissection proceeds to the lateral rim of the orbit, dissection should stop where the temporalis fascia splits into two layers and encases a fat pad. The outer fascial

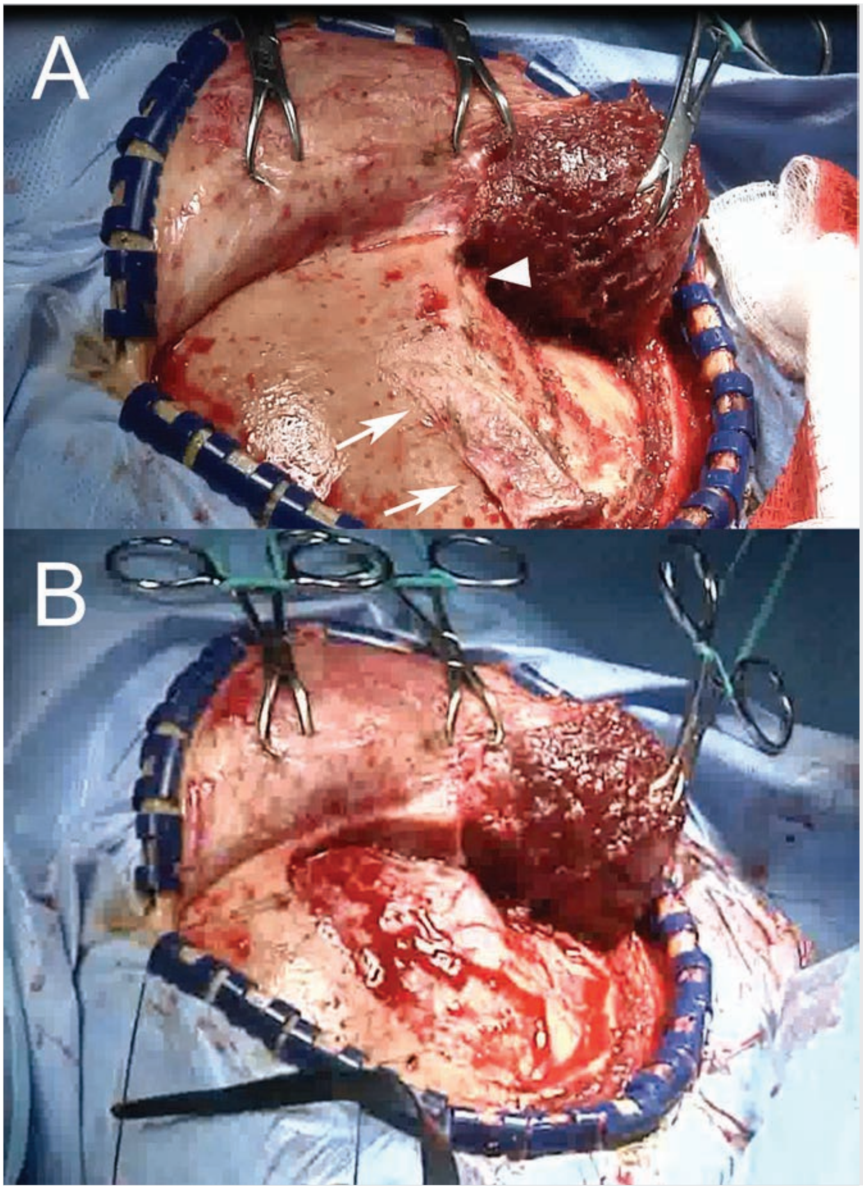

Fig. 4.2 **(a)** The scalp is reflected in one layer, leaving a segment of temporalis muscle and fascia on the bone (arrows) to suture the temporalis muscle to during the closure. The pterion and site for the keyhole burr hole (arrowhead) is exposed. **(b)** The craniotomy is done, the bone removed, and dural tack-up sutures are in place.

layer is incised here longitudinally and the fat dissected forward along with the scalp. This method allows the temporalis muscle to be reflected inferiorly, providing somewhat better exposure along the sphenoid ridge.

4.4.2 Craniotomy

The upper part of the radiolucent head holder is placed so that three fish hooks can be used to retract the scalp and muscle. If this is not available, towel clips and rubber bands can be used (▶ Fig. 4.2). The microscope and instruments for the intradural portion of the operation need to be prepared now so they are ready when needed. Burr holes are made. There are different options for burr holes. A single burr hole at the pterion or key hole can be adequate in a young person since the dura is not usually very adherent to the bone (▶ Fig. 4.2). This should be accurately placed below the most anterior part of the superior temporal line and above the frontozygomatic suture and drilled relatively perpendicular to the skull so as to avoid entering the orbit. Otherwise additional burr holes are made at the posteroinferior part of the exposure just superior to the root of the zygoma and at the posterior edge of the incision above the superior temporal line. The bone is cut out with a craniotome. The craniotomy can be adjusted depending on the location of the target aneurysm. For anterior communicating artery aneurysms, the frontal cut should be flush with or as close as possible to the roof of the orbit, extending medially to just before the supraorbital notch. For a middle cerebral artery aneurysm, this is less important, whereas bone removal should extend more into the temporal fossa.

The craniotome will usually not cut across the sphenoid ridge. The author uses a side-cutting attachment to cut a trough through the bone between the ends of each saw cut and then the bone can be elevated and the remaining small amount of bone of the sphenoid ridge will fracture and free the bone flap. Some bone removal along the sphenoid ridge can be done by dissecting the dura off the bone and drilling away the ridge or removing it with rongeurs. Bone removal required is variable and generally more for a posterior communicating or upper basilar artery aneurysms and less for a typical more superficial middle cerebral artery aneurysm.

Once the craniotomy is done, holes are drilled in the bone and sutures are placed in the dura to attach it to the bone (▶ Fig. 4.2). If holes are drilled in the bone flap for central tacking sutures, then work is completed with the drill and it can be moved away from the surgical field and kept sterile in the unlikely case that it is needed later. The dura can be opened in a **U** or **C** shape centered on the sphenoid ridge (▶ Fig. 4.3). It is retracted with 4–0 nonabsorbable sutures placed at the edges to keep it stretched. It is best not to open the dura until everything is set up for the intradural dissection including the loading of temporary clips so one is ready in the event of early intraoperative aneurysm rupture.

The dura and brain should be prevented from desiccation by putting cotton or similar slabs around the same way in every case, a narrow one temporally, then wide ones to cover the brain and make sure it and the dura do not dry out. This is the time to insert an EVD through Paine's point. Once the brain is covered, the microscope can be brought in to the field and the intradural operation performed.

4.4.3 Angiography

An intraoperative angiogram can be done through the superficial temporal artery and will result in good visualization of ipsilateral internal carotid and middle cerebral artery aneurysms and most anterior communicating artery aneurysms since the side of the approach will generally be from the side of the larger A1 (▶ Fig. 4.4, ▶ Fig. 4.5, ▶ Fig. 4.6).[3] The superficial temporal artery should be dissected free down to the level of the zygomatic arch. It is tortuous in about 80% of cases but becomes straighter inferior to the zygomatic arch. Tie it distally and under temporary clipping, make an arteriotomy, and insert an 18-gauge, 1.88-inch intravenous catheter, prefilled with heparinized saline and connected to a 4-inch extension tubing and 10-mL syringe. The artery is extremely prone to dissection and the catheter seems often to prefer to pass deep to the tunica intima. Suture the apparatus in place and it is ready for injection. The area of the craniotomy is covered with a towel and the intraoperative digital subtraction fluoroscopy can be brought in. Center the image on the clip, and have maximum magnification, all sponge radiopaque markers out of the way, and a projection that recreates one of the preoperative views that provides a good view of the aneurysm and surrounding arteries. Once this is set, flush the catheter with heparinized saline, switch to a syringe with 8 mL of intravascular contrast, have the anesthetist suspend respiration, and inject the contrast while recording in subtraction mode. The angiogram is not of perfect quality but one can see the aneurysm is gone and both A2 segments fill nicely (▶ Fig. 4.6 and **Video 4.1**).

4.4.4 Closure

Remove the catheter and securely ligate the superficial temporal artery proximal to the arteriotomy. The first dura stitch can be placed near the point in the durotomy half way from the frontal to the temporal end of the durotomy so as to match up the lengths and also get it stretched out properly (▶ Fig. 4.7). Do not let any blood get intracranially during closure. Try not to coagulate the bleeding edges of the dura too much as that can shrink it.

There are one or two central tacking sutures that are tied to the dura so they cannot be inadvertently pulled out. Irrigate at the highest point of the end of the dura closure to expel any subdural air. The bone can be put back with three small plates and screws. Someone can put those plates on as the dura is closed. Cosmetic results are important. Using a burr hole cover plate at the keyhole area may reduce the visible depression due to atrophy at the pterional area.

Now the temporalis muscle is sutured back to the cuff of muscle and fascia that was left on the superior temporal line

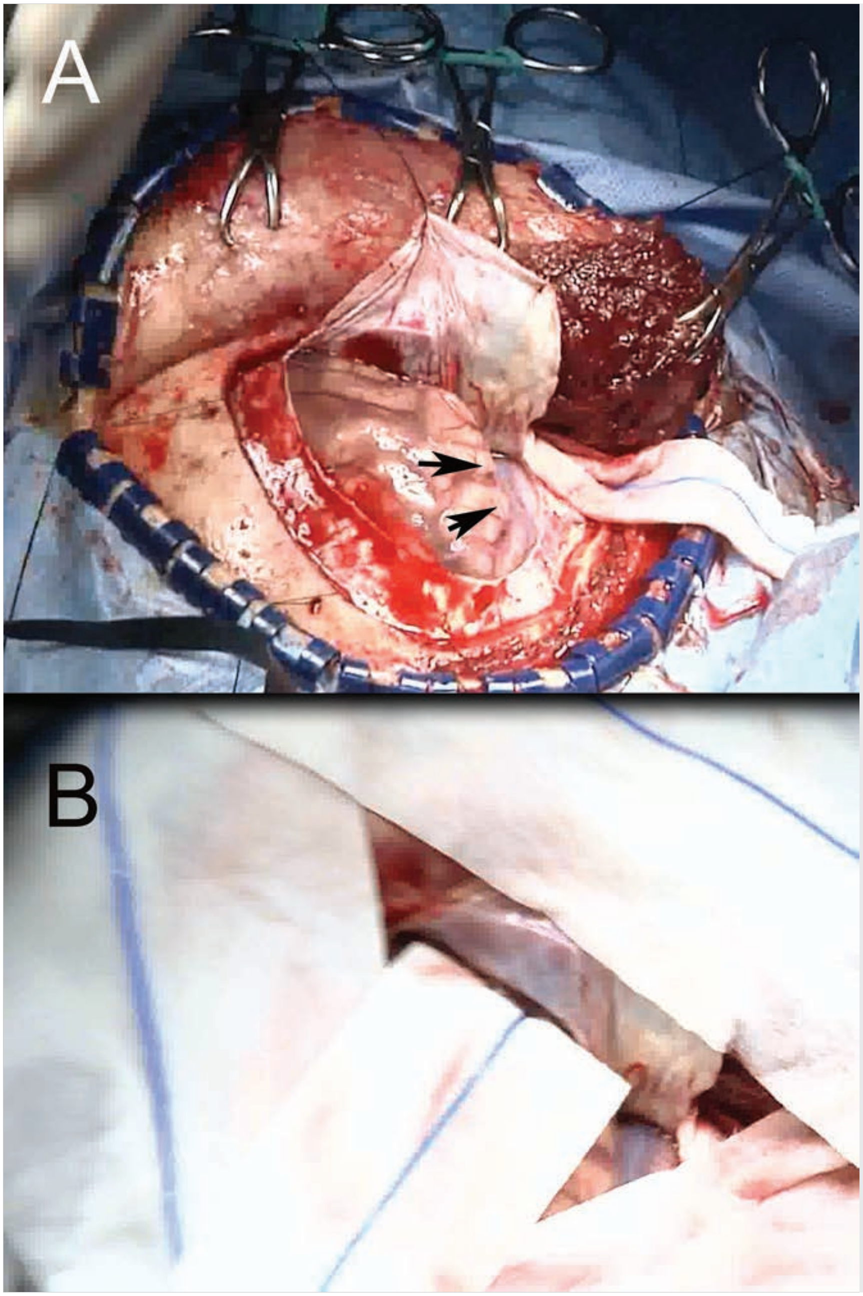

Fig. 4.3 **(a)** The dura is opened in a **U** or **C** shape based on the sphenoid ridge. It is retracted with two sutures placed at the cut edge of the dura so it remains under tension and does not shrink and make it more difficult to close the dura at the end of the intradural portion of the procedure. The sylvian fissure (*arrows*) is visible and there is relatively less temporal lobe exposure for this anterior communicating artery aneurysm surgery. A single narrow cottonoid is placed temporally. **(b)** The brain is covered with cottonoids so it does not dry out. The microscope can be brought in for the intradural portion of the operation.

with 3–0 absorbable sutures. Suture the fascia, and not the muscle, to minimize muscle atrophy. Close the galea with interrupted inverted 3–0 absorbable sutures, leaving the scalp clips on and removing them sequentially as the galea is sutured so as to reduce blood loss. The skin is always closed with a running subcuticular 4–0 Monocryl suture.

4.5 Postoperative Management Including Possible Complications

The patient is awakened and extubated if he/she was sufficiently alert to do so preoperatively and if there were no intraoperative catastrophes that might render the patient unsafe for extubation. Neurological examination is performed to detect any untoward effects of surgery. SAH patients are monitored in intensive care for fluid balance to maintain normovolemia and permissive hypertension. Any significant change in neurological condition warrants laboratory investigations and often an immediate computed tomography (CT) scan. We do not routinely perform cerebral angiography after clipping aneurysms but rely on intraoperative studies that are done in most cases, assuming they are adequate.

Complications or risks specifically of pterional craniotomy, excluding general complications (related to positioning and anesthesia and such) and those due to the intradural portions of the procedure the craniotomy is being done for since they are described in other chapters, include hemorrhage, wound/bone flap infection, frontalis nerve injury, and cosmetic issues.

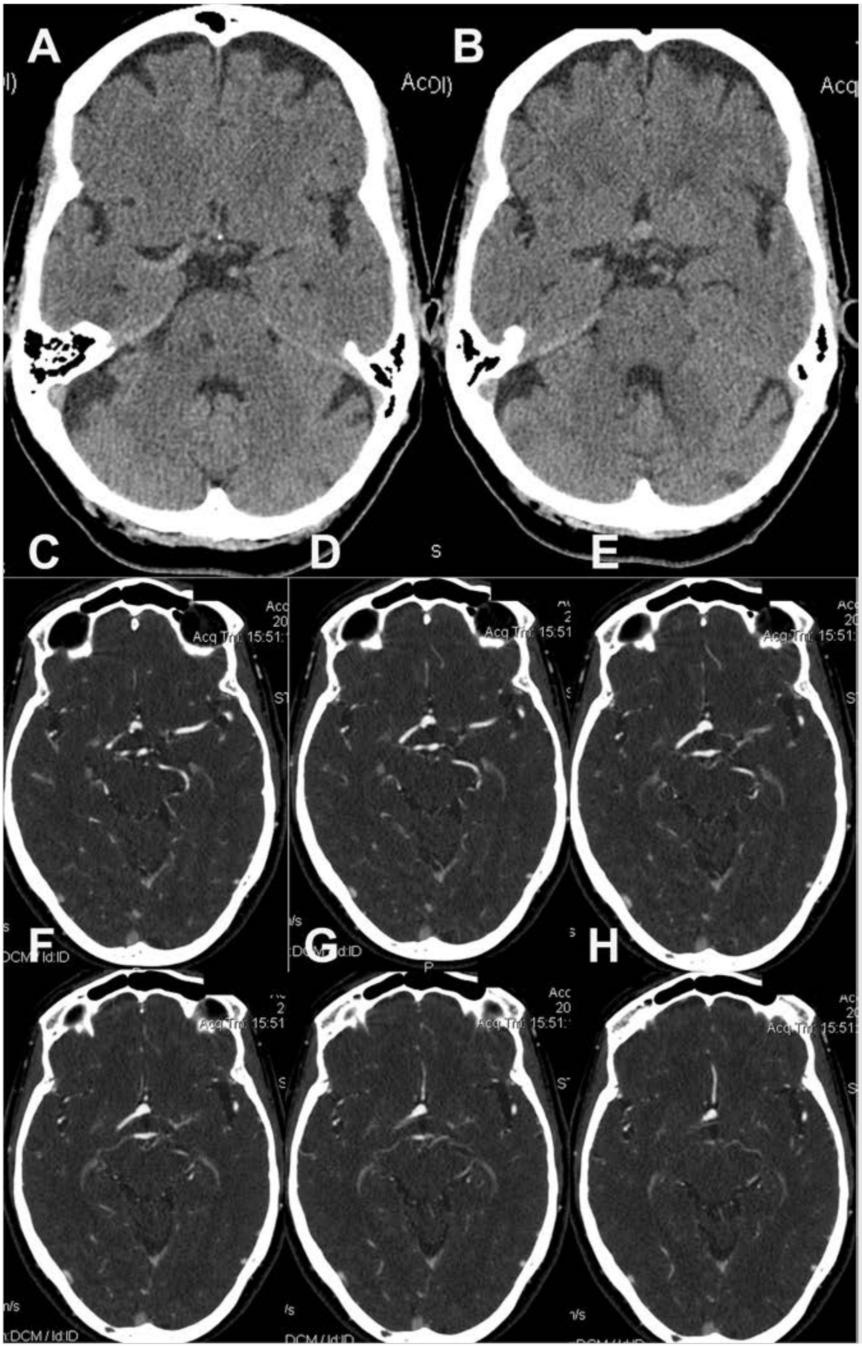

Fig. 4.4 **(a, b)** Preoperative plain computed tomography (CT) of a 66-year-old woman who complained of dizziness. **(c–h)** There is a 7-mm anterior communicating artery aneurysm that is delineated on axial views of a CT angiogram. The aneurysm arises at the right A1–A2 junction with a dominant right A1 and the aneurysm projecting to the left.

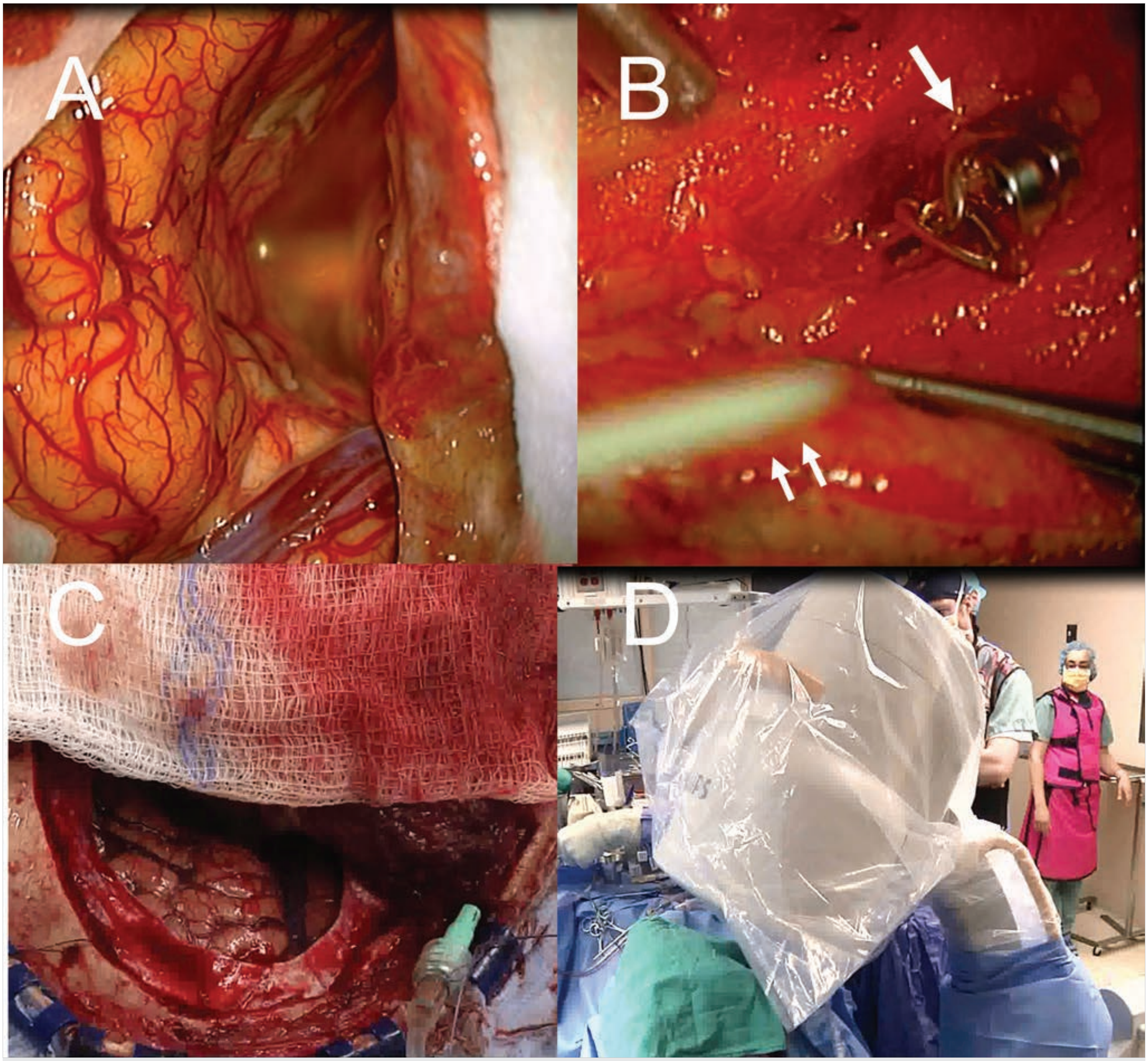

Fig. 4.5 (a) The brain at the end of the intradural surgery is slack and uninjured. **(b)** Cannulating the superficial temporal artery with temporary clip on the superficial temporal artery (*arrow*) and the 18-gauge intravenous catheter about to be inserted into the arteriotomy (*arrows*). **(c)** The catheter is connected to a short extension tube and a 10-mL syringe. **(d)** The room setup for intraoperative angiography through the superficial temporal artery.

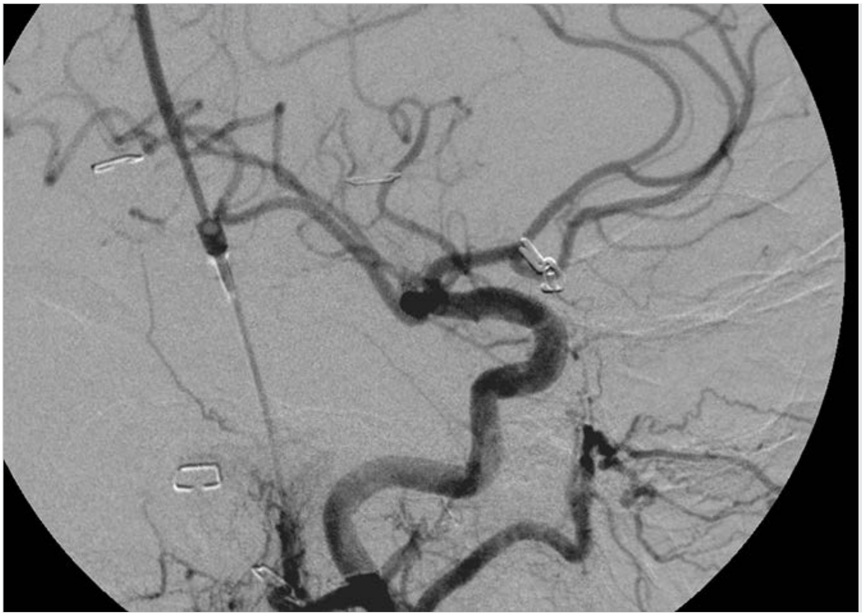

Fig. 4.6 Intraoperative angiogram shows occlusion of the aneurysm and patency of right A1 and both A2 segments.

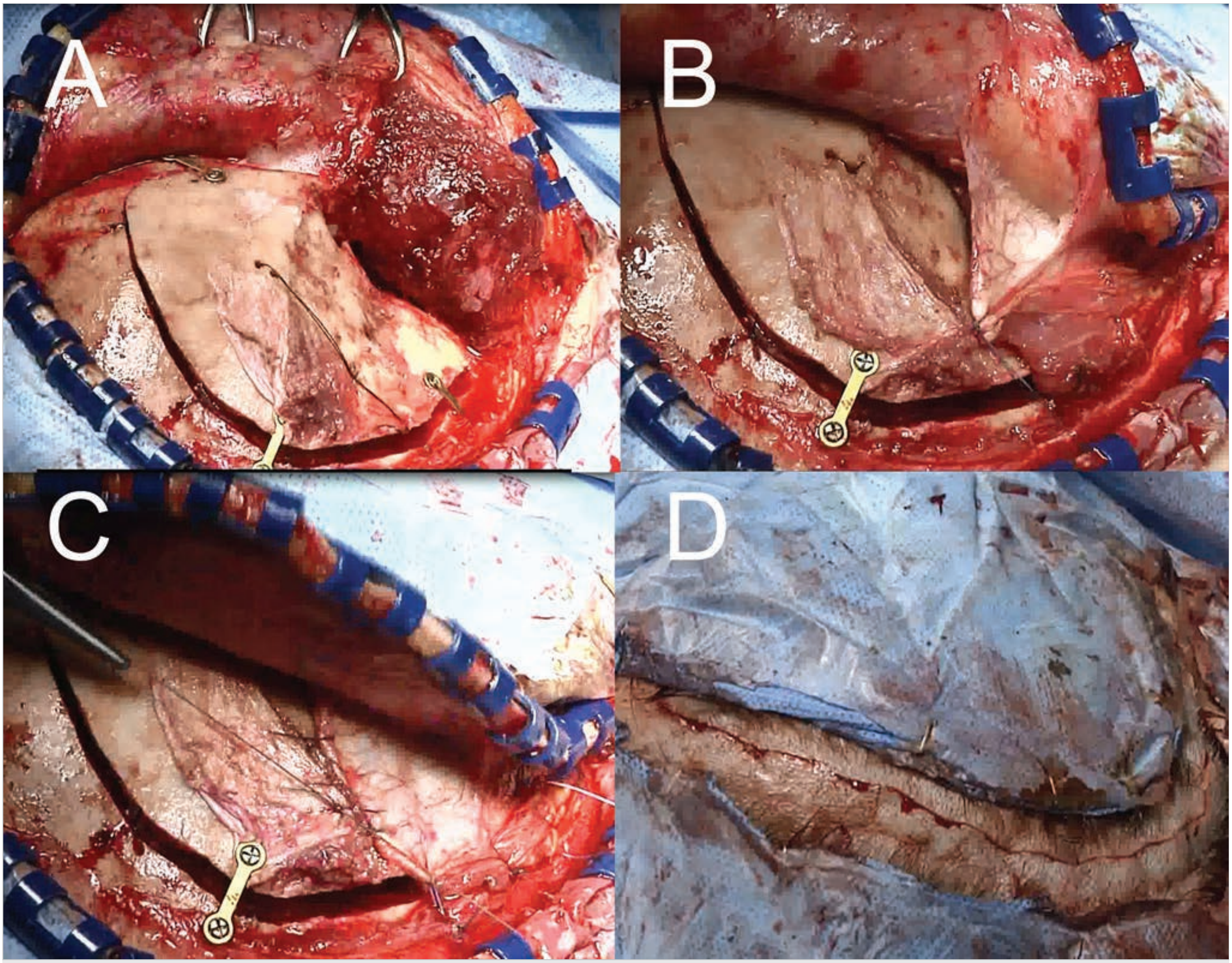

Fig. 4.7 **(a)** The bone is fixed in place with three titanium plates. The central tacking suture can be seen emanating through two holes in the bone just above the superior temporal line. **(b)** The temporalis fascia is sutured back to the cuff of muscle and fascia that was left attached to the bone. **(c)** Further suturing of the temporalis fascia. **(d)** Intradermal skin closure will provide a good cosmetic result for the incision and alleviate any patient anxiety about having to have staples removed.

Dural tack-up sutures and sutures tying the dura to the middle of the bone flap help reduce the risk of postoperative epidural bleeding. Superficial wound infections can be treated initially with antibiotics but this will not generally cure a deeper infection of the bone, for which the bone flap should be removed. Resorption of the bone flap over weeks to months can be a sign of infection. The frontalis nerve can be injured by stretch or surgical division. A frontalis branch palsy is most likely to be permanent.

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5 Minimally Invasive Approaches to Aneurysms

Hans-Jakob Steiger and Daniel Hänggi

Abstract

Minimally invasive approaches for aneurysms have potential advantages over traditional craniotomies, such as shorter operation times and less scalp and muscle dissection, potentially resulting in less postoperative pain and in improved cosmetic results. On the other hand, there are unique requirements for their use, including adequate preoperative imaging to ensure the aneurysm is appropriate for such an approach, good technical traditional microsurgical skills, and appropriate instrumentation for the procedure. This chapter describes the selection of patients and techniques for common minimally invasive approaches.

Keywords: cerebral aneurysm, craniotomy, minimally invasive

5.1 Patient Selection

The minimally invasive approach for aneurysms means small and strategically placed craniotomies (▶ Fig. 5.1), avoiding brain retraction and minimizing tissue damage during dissection. Acceptable minimally invasive approaches must provide sufficient control for ruptured as well as unruptured aneurysms. We do not encourage using very small openings for unruptured aneurysms that only allow clipping at the neck of the aneurysm and do not permit adequate proximal and distal control. In the event of an intraoperative rupture, such minimal-access procedures are a setup for an adverse outcome. Also, the suspected difficulty of clipping the aneurysm needs to be considered. Features such as larger aneurysm size, calcification and thrombus in the parent artery or aneurysm, and perceived need for bypass or other complex reconstruction techniques will weight against employing a minimally invasive craniotomy.

The critical aspect of these craniotomies is to use three-dimensional imaging to define the relation between the skull (entry point) and the position of the aneurysm (target point) and to select the appropriate craniotomy based on this plan (▶ Fig. 5.2 and ▶ Fig. 5.3).

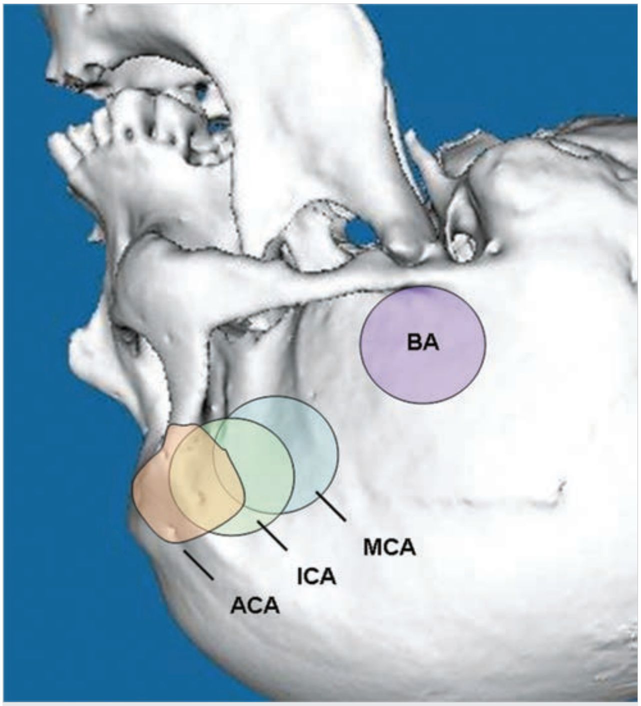

Fig. 5.1 Minimal invasive keyhole craniotomies to the typical aneurysm sites. The specific minimally invasive craniotomies are designed to provide adequate exposure and control for the specific aneurysms instead of a universal aneurysm craniotomy, such as a generous pterional craniotomy. ACA, orbitocranial opening for anterior communicating artery aneurysms; BA, subtemporal keyhole approach to basilar bifurcation and basilar superior cerebellar artery aneurysms; ICA, minipterional craniotomy for internal carotid artery aneurysms; MCA, sylvian craniotomy for middle cerebral artery aneurysms.

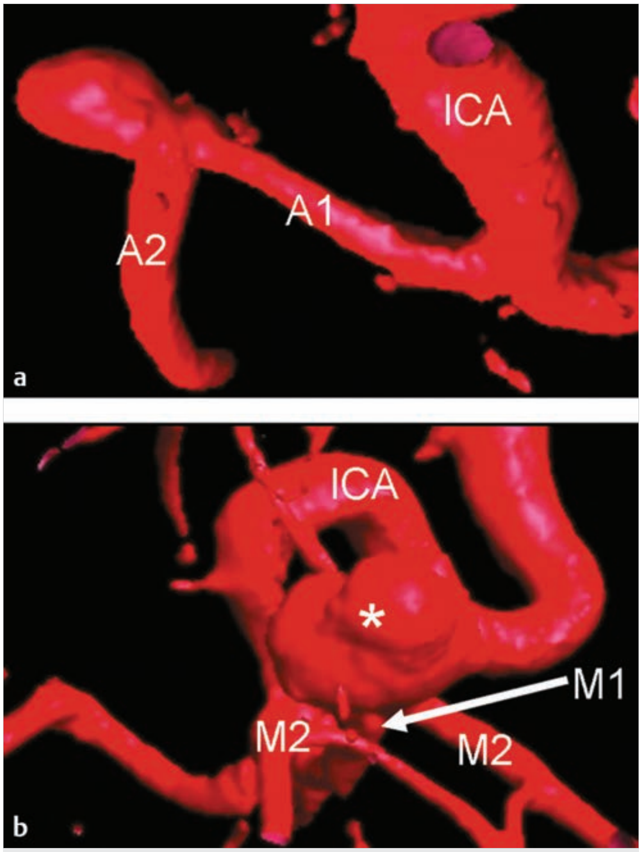

Fig. 5.2 Three-dimensional rotational digital subtraction angiography is helpful to plan minimally invasive approaches to specific aneurysms. The reconstruction allows planning the approach and clip application as well as clip selection. **(a)** 3D overview of a right middle cerebral artery aneurysm. **(b)** Higher magnification of the right MCA bifurcation. Proximal control of the sphenoidal segment (M1) of the middle cerebral artery is achieved either between the middle cerebral branches (M2) or medial to the superior trunk, depending on the course of the M1 and M2 branches.

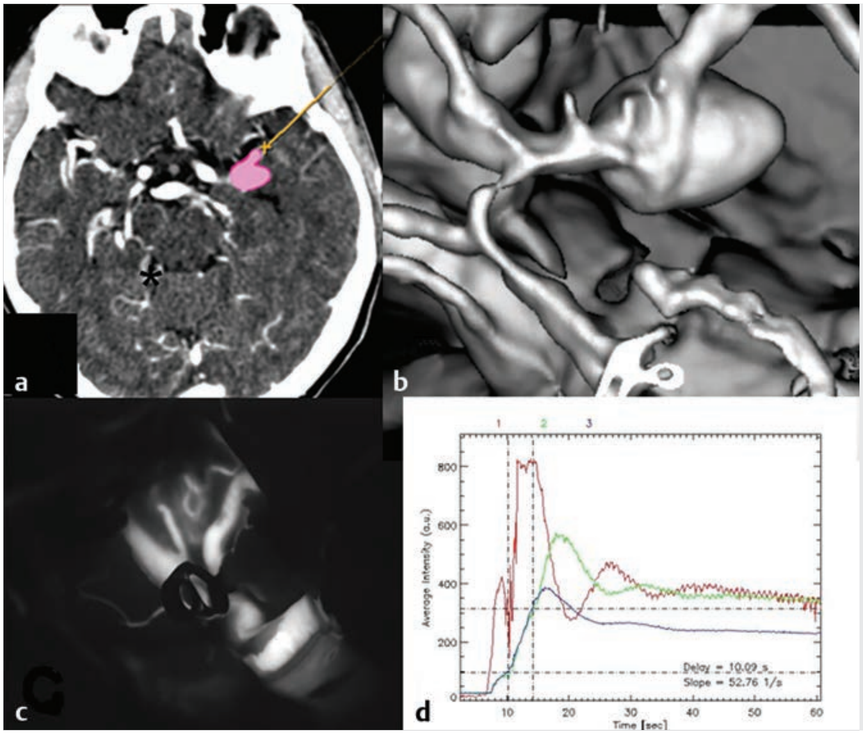

Fig. 5.3 Recent technological innovation has made aneurysm surgery easier, safer, and more precise. **(a, b)** Neuronavigation is helpful for middle cerebral artery (MCA), pericallosal artery, and peripheral aneurysms. **(c)** Indocyanine green videoangiography has become an accepted tool for intraoperative assessment of parent artery patency, **(d)** Quantified measurement of fluorescence intensity (Flow 800, Zeiss) with differential analysis of transit times in arteries, parenchyma, and veins still must be considered experimental.

5.2 Preoperative Preparation

Prior to surgery, a master plan of the craniotomy and approach to the aneurysm should be made. The most direct route to the aneurysm that requires minimal retraction and microsurgical dissection should be identified. We distinguish aneurysms accessible through basal approaches (aneurysms of the carotid artery) and those accessible through hemispheric approaches (middle cerebral artery [MCA] and pericallosal artery aneurysms). Anatomical landmarks are adequate for placement of the craniotomy for aneurysms near the skull base, whereas image guidance is useful for hemispheric approaches.

Brain retraction should be minimized by achieving brain relaxation with cisternal, ventricular, or spinal drainage of cerebrospinal fluid and by administering mannitol if necessary. We use a spinal or ventricular catheter for all operations in the acute stage after subarachnoid hemorrhage (SAH). Lumbar drainage is preferred for good-grade patients and ventricular drainage for the worse grades.

5.3 Operative Procedure

5.3.1 Keyhole Approach to Middle Cerebral Artery Aneurysms

Craniotomies for MCA aneurysms can be divided into frontolateral and temporal approaches and then by whether one approaches the aneurysm by following the proximal MCA (M1) from the internal carotid artery (ICA) bifurcation or inward from the sylvian fissure. Controlling M1 from the ICA bifurcation appears safer, but accessing M1 close to the ICA bifurcation may require considerable retraction of the fronto-orbital cortex and also maneuvering around the aneurysm. Also, particularly in cases with a long M1, this artery tends to course high in its middle segment before turning downward again at the bifurcation. We prefer to control M1 by following the superior trunk (M2) of the MCA proximally to the M1. Depending on the exact anatomy of the bifurcation and aneurysm, M1 is controlled either between the MCA main branches posteriorly or on the frontal side of the superior M2 (▶ Fig. 5.2). In cases with a temporal intracerebral hematoma, we initially evacuate the hematoma via a corticotomy in the superior temporal gyrus. We then expose the MCA bifurcation through the hematoma cavity or through the sylvian fissure.

For most MCA aneurysms, the head is positioned with a 45-degree rotation to the contralateral side. A limited hairline incision is fashioned that allows sufficient exposure down to the orbital rim. The temporal muscle is incised and the anterior portion of the muscle is mobilized together with the skin flap. A round craniotomy 3 cm in diameter is placed on the sylvian fissure, immediately behind the orbital rim (▶ Fig. 5.1). Two-thirds of the craniotomy should lie above the sylvian fissure and one-third below. The dura is opened as a flap with an anterolateral base.

The sylvian vein is identified. The lumbar or ventricular catheter is opened to drain cerebrospinal fluid and relax the brain. If relaxation is insufficient, mannitol is given (1 g/kg body weight). If there is a large intracerebral hematoma, sufficient relaxation is achieved by evacuation of the hematoma. The sylvian fissure is split on the frontal side of the sylvian vein. Venous branches crossing the fissure must usually be coagulated and divided. The superior trunk of the MCA is then identified in the depth of the sylvian fissure and followed proximally to the MCA bifurcation. For proximal control, it is important to have the course of the M1 and the projection of the aneurysm in mind and to dissect along the side opposite the projection of the aneurysm (▶ Fig. 5.2).

We do not use brain retractors for MCA aneurysms unless the brain remains full despite sufficient brain relaxation. Clipping the aneurysm does not differ with the limited approach. We use temporary clipping when the neck of the aneurysm is wider than the diameter of the M1. We keep the blood pressure at a normal level and do not use pharmacological neuroprotection. Closure of the craniotomy is unremarkable. We do not use wound drains.

5.3.2 Transorbital Keyhole Approach to Anterior Communicating Artery Aneurysms

We introduced the transorbital keyhole approach as a more ventral means of access to the anterior communicating artery complex, which avoids retraction of the orbital cortex and resection of the gyrus rectus (▶ Fig. 5.4). The goals are to have a sufficiently wide exposure to allow for unconfined manipulation and control of the relevant parent arteries but with a minimal size. The approach is from the side of the dominant precommunicating anterior cerebral artery (A1). The head is positioned in 45-degree rotation to the contralateral side and with 10-degree hyperextension to allow the orbital cortex to fall away from the skull base.

We use a frontotemporal hairline incision, although the eyebrow incision is an alternative. The skin incision is started 5 mm in front of the tragus, slightly above the zygomatic arch. The scalp incision is extended to the frontal midline. The skin flap is reflected in the interfascial plane between the temporal fascia and the aponeurosis of the temporal muscle, mobilizing the supraorbital fat pad to prevent injury to the frontal branch of the facial nerve. The scalp flap is retracted basally. The anterior attachment of the temporal muscle at the orbital pillar and the anterior aspect of the linea temporalis are incised sharply. The temporal muscle is then dissected from the underlining bone and pulled back. The lateral and superior orbital rims are dissected from the periorbita using a blunt curved dissector. Attention should be paid to the frontal nerve as it turns around the orbital rim at the medial aspect of the planned craniotomy. Occasionally, it may be necessary to mobilize the frontal nerve out of its bony groove or tunnel.

The craniotomy has two burr holes. The first is at the keyhole (▶ Fig. 5.4). The direction of drilling, however, aims more toward the orbit so that the orbit and the anterior cranial fossa are opened simultaneously. The second burr hole is at the medial aspect of the craniotomy immediately above the orbital rim. The burr holes are connected with the craniotome along

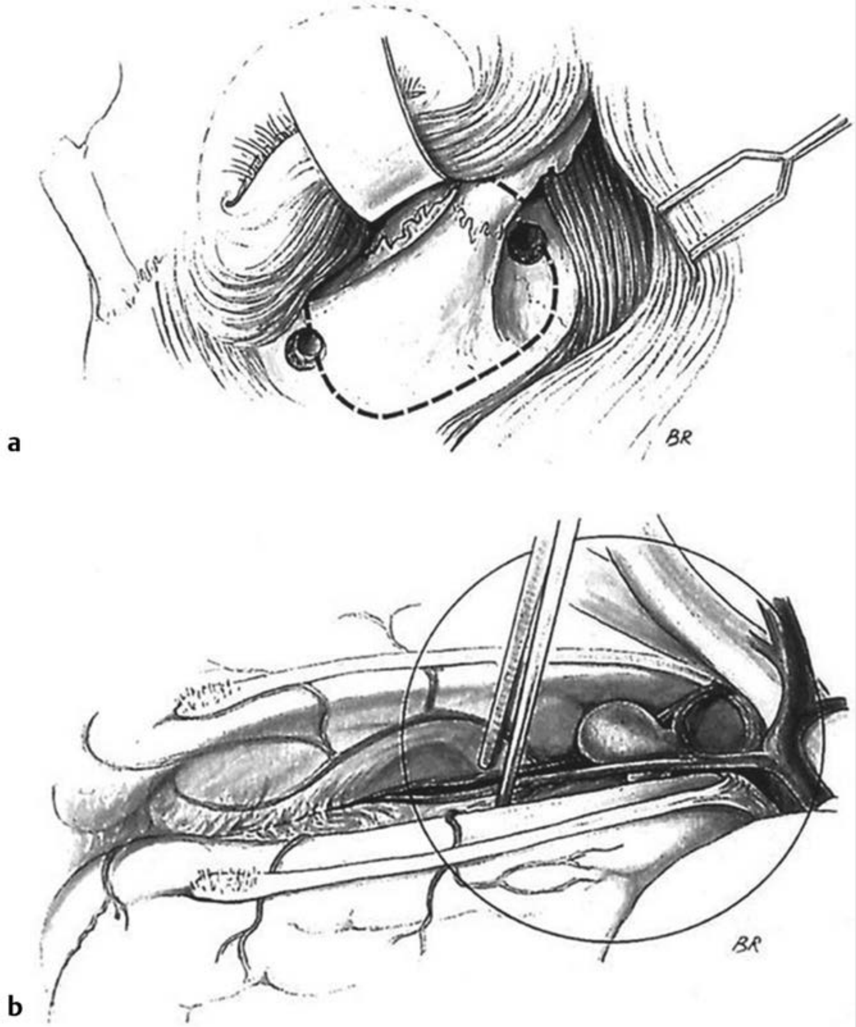

Fig. 5.4 Transorbital keyhole craniotomy for anterior communicating artery aneurysm. This approach requires minimal brain retraction and allows approaching the aneurysm through the ventral interhemispheric fissure. **(a)** Orbital craniotomy including the orbital rim and orbital roof. **(b)** Dissection of the ventral interhemispheric fissure and exposure of the aneurysm. (Reproduced with permission from Steiger HJ, Schmid-Elsaesser R, Stummer W, Uhl E. Transorbital keyhole approach to anterior communicating artery aneurysms. Neurosurgery 2001;48:347–351.)

the upper delineation of the planned opening. The superior and lateral orbital rims are best cut with a bone saw. The dura is protected with a spatula. The bone cut in the lateral orbital rim is extended down to the lateral burr hole. With the use of a small punch inserted into the lateral burr hole, the orbital roof is divided along the pterion to a depth of 3 to 4 cm. The anterior aspect of the medial orbital roof is divided through the medial burr hole with a small punch or diamond drill. The bone flap is then elevated and the posterior orbital roof is fractured between the lateral and medial orbital roof cuts.

The dura is opened with a base inferiorly. Brain relaxation is achieved as already described. The orbital cortex is elevated and the sylvian fissure is identified at the lateral aspect of the exposure. The fissure is split down to the ICA bifurcation. The posterior aspect of the gyrus rectus is now separated from the chiasm. The A1 segment of the anterior cerebral artery is usually visible for proximal control at this stage. The next step is to split the interhemispheric fissure (▶ Fig. 5.4). The projection of the aneurysm dome must be kept in mind at this stage. The posterior aspect of the gyrus rectus is completely freed at this point and can be mobilized upward to expose the A1–A2 junction. The exposure is stabilized with a small brain spatula. To distribute the pressure on the orbital cortex, it is helpful to insert a rolled cottonoid in front of the spatula between the orbital roof and orbital cortex. During this maneuver, attention must be paid to the olfactory bulb.

A straight anterior projection of the aneurysm dome may render formal splitting of the interhemispheric fissure dangerous. In these situations, it may be more appropriate to perform a small resection of the gyrus rectus medial to the olfactory tract.

Following clipping of the aneurysm, the dura is closed in a watertight fashion. If the frontal sinus has been opened, the defect is plugged with a muscle graft from the temporal muscle. The bone flap is secured with rivets or microplates. The anterior aspect of the temporalis muscle is reattached at the linea temporalis by making small holes in the bone flap and securing it with unresorbable 3–0 sutures.

5.3.3 Minipterional Craniotomy for Internal Carotid Artery Aneurysms

The keyhole approach to ICA aneurysms does not vary from the standard pterional craniotomy other than in size. The head is positioned at a 45-degree angle toward the opposite side and hyperextended 10 degrees to facilitate separation of the brain from the base of the skull. We use the same frontotemporal scalp/muscle flap as for MCA aneurysms. The medial aspect of the skin incision must extend a little more toward the midline.

The central point of reference for the craniotomy and the first burr hole is at the frontozygomatic suture at the keyhole. The relative extension forward and back and the exact positioning of the head depend on the specific aneurysm location. For an ophthalmic artery aneurysm, no or only minimal hyperextension is necessary, whereas substantial hyperextension is an advantage for ICA bifurcation aneurysms. One or more additional burr holes can be placed. The bone flap measures 3 cm in diameter. On the temporal side, the exposure should cross the sylvian fissure, and on the frontal side it should go down to the orbital roof. Additional area is gained basally by removing the sphenoid ridge with rongeurs or a diamond drill.

The dura is opened basally in an arc-shaped fashion, crossing the sylvian fissure. The sylvian fissure is split starting laterally on the frontal side of the sylvian vein. Spreading movements with the bipolar tweezers are sufficient to open the arachnoid. Brain relaxation is achieved by maneuvers described earlier. After the aneurysm is clipped, the dura is closed in a watertight manner and the bone flap is replaced. The mobilized part of the temporal muscle is fixed at the linea temporalis of the skull with two unresorbable sutures that are pulled through tangentially fashioned drill holes.

5.3.4 Subtemporal Keyhole Approach to Upper Basilar Artery Aneurysms

This approach was developed by Drake and was probably the first keyhole approach that found wide acceptance (▶ Fig. 5.5). A disadvantage of the subtemporal approach is the high rate of temporary oculomotor nerve dysfunction, although this usually resolves in 6 to 8 weeks. The limitation of the subtemporal approach is that it is not suitable for high basilar bifurcations (more than 10 mm above the dorsum sellae). The relation of the aneurysm neck to the dorsum sellae must be studied on the preoperative angiography. The approach is also unsuitable for low basilar bifurcations (5 mm or more below the dorsum sellae). The scope of the approach can be extended inferiorly by removing the posterior clinoid process or the petrous apex. These extensions of the approach are associated with additional risk.

The typical basilar apex aneurysm can be accessed along anatomical landmarks. For more peripheral aneurysms, such as posterior cerebral or peripheral superior cerebellar artery aneurysms, neuronavigation is recommended. In contrast to all other aneurysm locations, we monitor somatosensory and motor evoked potentials for basilar artery aneurysms.

For the subtemporal keyhole approach, the patient should be positioned in a lateral decubitus or park bench position. A supine position with the shoulder supported is less optimal because it imposes more stress on the cervical spine, and venous drainage from the head is more likely to be impeded. The head should be secured in rigid pin fixation in a horizontal position and hyperextended 10 degrees as with the other basal approaches described. Normally, an approach from the nondominant hemisphere is preferred. If the basilar apex is asymmetric, however, we prefer the side with the shorter distance to the aneurysm. Basilar superior cerebellar artery aneurysms must be approached from the side of the aneurysm projection.

A linear preauricular 10-cm-long skin and muscle incision is fashioned. The incision starts from the root of the zygomatic process and should be placed not more than 5 mm in front of the tragus to avoid damage to the frontal branch of the facial nerve. The split temporal muscle must be separated from the squama temporalis. In general, the center of the craniotomy must be slightly in front of the skin incision. The root of the processus zygomaticus corresponds approximately to the projection of the basilar bifurcation and should be taken as a landmark to place the craniotomy. We use a small osteoplastic

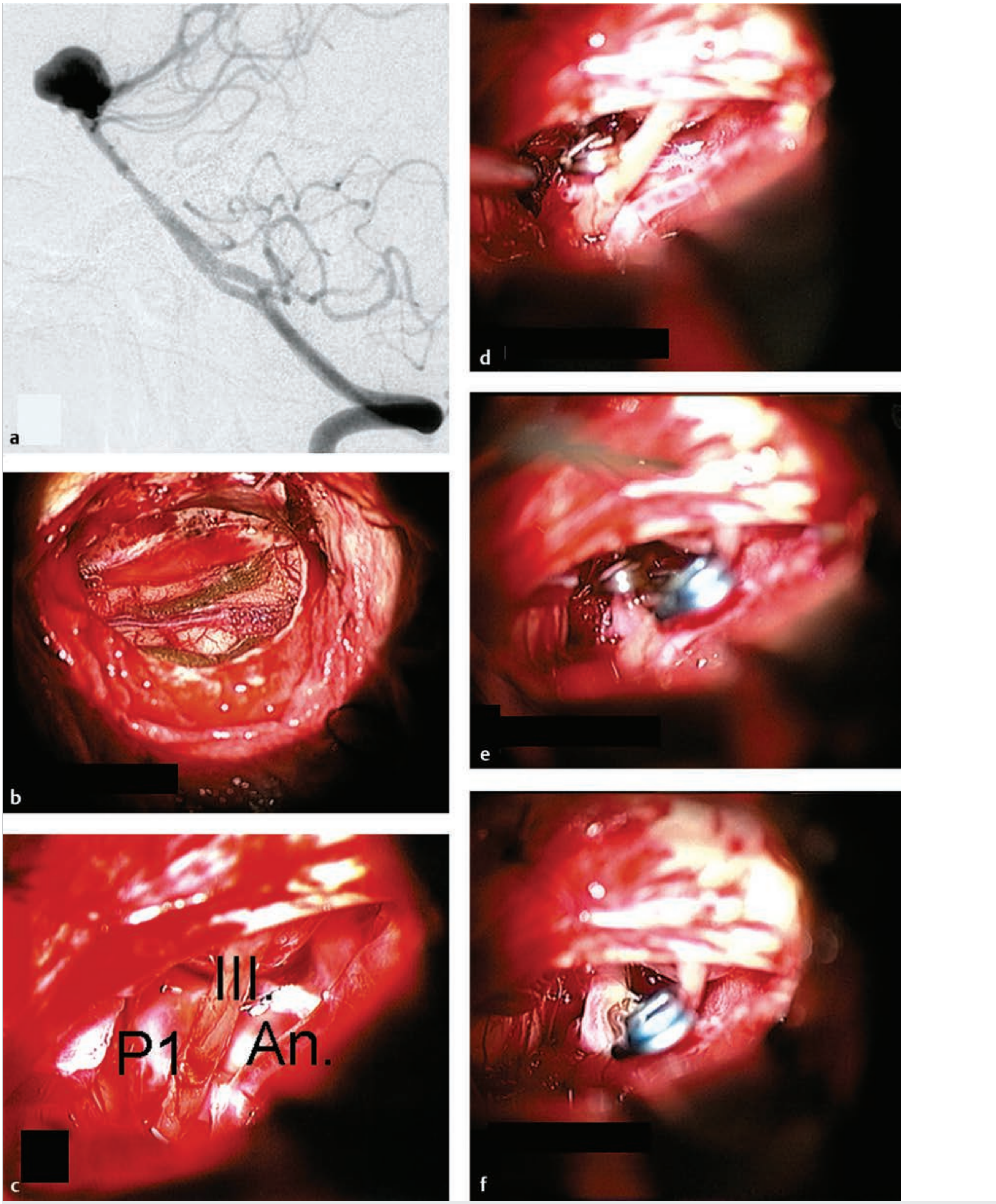

Fig. 5.5 Left subtemporal keyhole approach to a wide-based basilar bifurcation aneurysm. **(a)** Lateral projection of digital subtraction angiography. **(b)** 3- to 4-cm subtemporal craniotomy and dural opening. **(c)** The basilar apex and the aneurysm after exposition and dissection. The tentorial edge has been retracted to the floor of the middle fossa with a suture (III, third cranial nerve; P1, left posterior cerebral artery; An, aneurysm). **(d)** Temporary clipping of the basilar artery. **(e)** Insertion of the aneurysm clip in front of the left P1. **(f)** Clipped aneurysm after removal of the temporary clip. Careful inspection of the clip branches after clip application is crucial with these aneurysms to prevent occlusion of critical perforators.

craniotomy 3 to 4 cm in diameter. The craniotomy must extend to the base of the middle cranial fossa.

The dura is opened with a basally pediculated flap. Brain relaxation is achieved by cerebrospinal fluid drainage and mannitol. Ventricular or lumbar drainage, however, is mandatory for the subtemporal approach. The subtemporal surface of the brain is now inspected. The vein of Labbé must be identified and protected. The subtemporal cortex is then freed from the floor of the middle cranial fossa. Some small veins may need to be coagulated and divided. The temporal lobe is protected with Gelfoam (Pfizer Inc., New York, NY) or comparable material. Next the brain is elevated off the middle fossa floor and stabilized with two spatulas. Rolled cottonoids can be inserted between the skull base and the brain on both sides of the spatulas to distribute the force and to avoid retraction damage. Forceful retraction must be absolutely avoided.

The tentorial edge is identified. To increase the working space, we use the Drake method and suture the tentorial edge down to the dura of the middle fossa. The basilar artery, its branches, and the neck of the aneurysm are then dissected. After clipping of the aneurysm, the dura and craniotomy wound are closed in the usual manner.

5.4 Postoperative Management Including Complications

The small craniotomies described in this chapter do not require any particular postoperative measures. Wound drains are not required. We recommend postoperative angiography 5 to 7 days after SAH. This time allows identification of vasospasm prior to development of irreversible infarctions and therefore a chance for corrective measures such as angioplasty. Control angiography may also be justified to document adequate exclusion of the aneurysm.

In summary, minimally invasive approaches for aneurysm surgery are a necessary development in the light of the less invasive endovascular therapies. Minimally invasive access is achieved by eliminating the unnecessary parts of the traditional craniotomies and intradural manipulations. Despite smaller openings, a minimally invasive access must always provide sufficient working space for safe control of the aneurysm and the parent arteries. The operative field must never be narrow and confining. Neuronavigation is a useful adjunct for planning the approach to atypical and peripheral aneurysms.

6 Ophthalmic Segment Aneurysms

Arthur L. Day and Ali Hassoun Turkmani

Abstract
The surgical treatment of ophthalmic segment aneurysms is challenging because of their proximity to the anterior skull base and the visual system. Unlike other anterior circulation aneurysms, the treatment of these lesions usually requires extensive removal of the anterior clinoid process and may require cervical carotid exposure for proximal control. The advent of endovascular neurosurgery and the development of new endoluminal flow-diverting devices have led to an important paradigm shift in the management of many ophthalmic segment aneurysms. By adapting Rhoton's "aneurysm rules" and knowing the specific surgical anatomy of this region, microsurgical treatment remains a viable alternative and often preferential method of management for many of these lesions.

Keywords: cerebral aneurysm, carotid artery, ophthalmic artery, microsurgery

6.1 Aneurysm Anatomic Analysis and Indications for Surgical Intervention

The gold standard for aneurysm diagnosis continues to be a complete transfemoral digital subtraction angiogram. The catheter angiogram will also show any associated vascular abnormalities (i.e., arteriosclerosis with luminal narrowing) that may influence the treatment method. The size and shape of the aneurysm, its direction of projection, and its appearance in relation to the size of the lesion on computed tomography (CT) or magnetic resonance imaging (MRI) should be noted.[1] A lesion on CT or MRI larger than one seen on angiography suggests intra-aneurysmal thrombosis. CT angiography in multiple planes can help discern whether the neck of the aneurysm is intra-, inter-, or extradural. Calcification best noted on the CT scan can make clipping more difficult.[2]

6.1.1 Anatomy of the Ophthalmic Segment

The ophthalmic segment (OphSeg) is that portion of the internal carotid artery (ICA) that extends from the point the ICA enters the subarachnoid space to the origin of, but not including, the posterior communicating artery (PComArt). The OphSeg is preceded by the cavernous and clinoidal segments of the ICA.[3] The cavernous segment (CavSeg) lies within the dural confines and venous lumen of the cavernous sinus, outside the subarachnoid space. As the CavSeg ascends just beyond its anterior genu, it pierces the carotid-oculomotor membrane and becomes the clinoidal segment (ClinSeg).[4] The ClinSeg is covered laterally by the anterior clinoid process (ACP) and ascends and passes through the dura at the dural ring (DR) to enter the subarachnoid space, at which point it becomes the OphSeg (▶ Fig. 6.1 and ▶ Fig. 6.2).[5,6]

OphSeg aneurysms are defined by their relation to the two named branches of the segment, which are the ophthalmic and superior hypophyseal arteries.[6] The ophthalmic artery (OphArt) arises from the dorsal or dorsomedial aspect of the ICA just distal to the DR and courses anteriorly through the optic canal underneath the lateral aspect of the optic nerve.[7] The first of often multiple superior hypophyseal arteries (SupHypArt) originates just distal to the DR as one or several perforators from the medial or inferomedial aspect of the ICA.[8]

6.1.2 Classification of Ophthalmic Segment Aneurysms

Three aneurysm variants arise with the OphSeg (▶ Fig. 6.3).[9,10] *OphArt aneurysms* arise from the ICA just distal to the origin of the OphArt and project dorsally or dorsomedially toward the lateral aspect of the optic nerve. *SupHypArt aneurysms* arise along the medial surface of the ICA in association with one or several perforators from the medial or inferomedial ICA wall that supply the optic chiasm and pituitary stalk. Some project inferomedially toward the sella turcica and burrow into a diverticulum of the subarachnoid space medial to the ICA (the carotid cave), and are termed the parasellar variant.[11] Others extend early in their growth directly into the suprasellar space and represent the suprasellar variant.[9] Both variants are more difficult to treat surgically than OphArt types because of their medial projection and proximal origins.[12,13] The much less common *dorsal variant OphSeg aneurysm* arises several millimeters distal to the origin of the OphArt and projects superiorly. Some may be typical saccular aneurysms, but others appear as "blisters" on the dorsal surface of the ICA.[14]

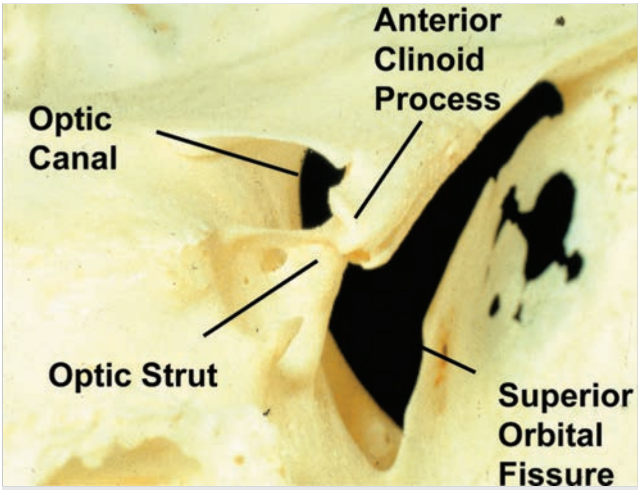

Fig. 6.1 Osseous anatomy. The anterior clinoid process is the medial extent of the lesser sphenoid wing and is connected on its inferomedial aspect to the sphenoid bone by the optic strut. The anterior clinoid process together with the optic strut forms the lateral and inferior wall of the optic canal.

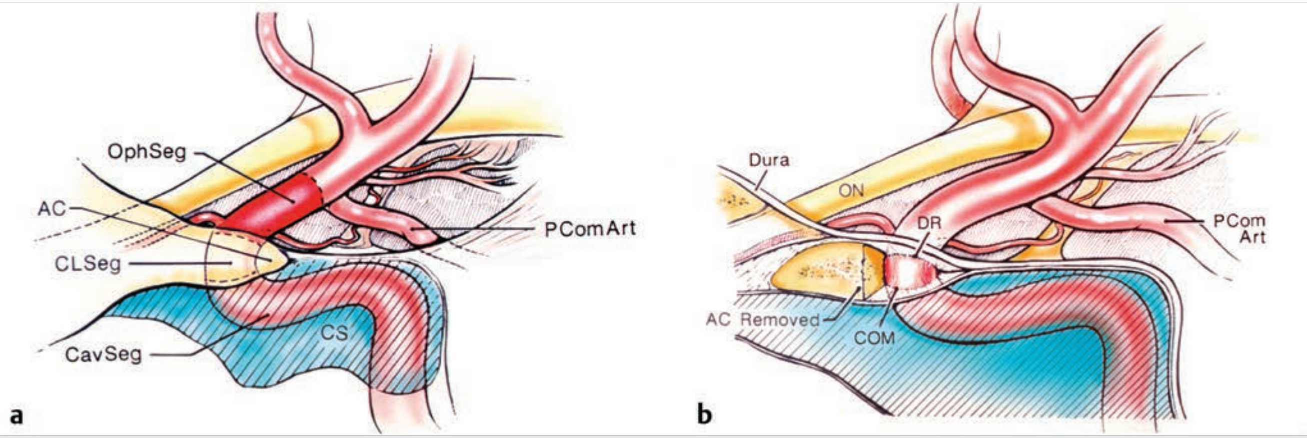

Fig. 6.2 OphSeg anatomy. **(a)** Lateral view with the clinoid intact. The segments of the internal carotid artery can be identified, including the intracavernous segment (CavSeg), the clinoidal segment (CLSeg) covered by the anterior clinoid process (AC), and the ophthalmic segment (OphSeg). **(b)** Lateral view with the anterior clinoid process removed shows that the OphSeg begins at the dural ring (DR) and ends at the origin of the posterior communicating artery (PComArt). COM, carotid-oculomotor membrane; CS, cavernous sinus; ON, optic nerve.

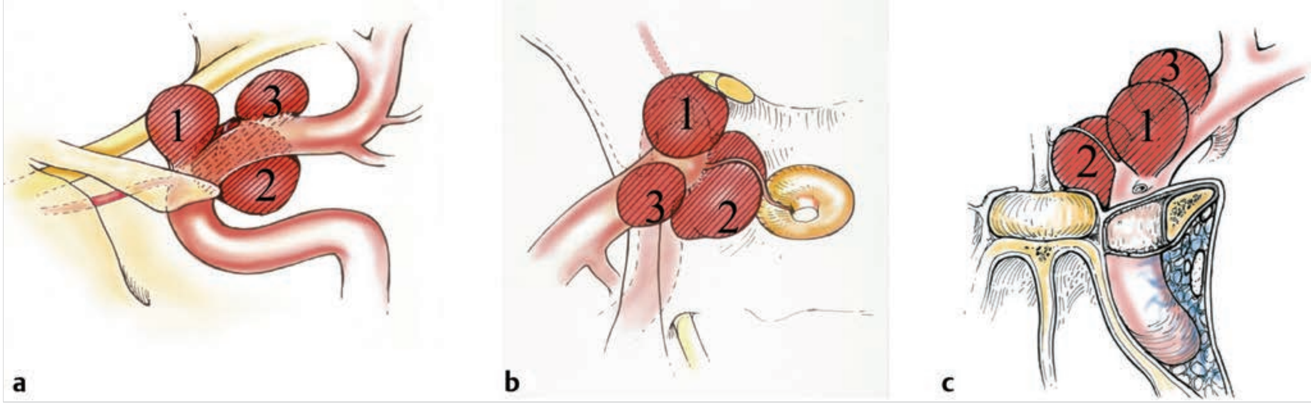

Fig. 6.3 Typical anatomy of OphSeg aneurysms from **(a)** lateral, **(b)** superior, and **(c)** anterior views. The OphArt aneurysm (1) typically arises from the dorsal surface of the internal carotid artery (ICA) just distal to the OphArt and projects dorsally or dorsomedially. The SupHypArt aneurysm (2) arises from the inferomedial surface of the ICA and projects medially usually into the carotid cave. The rarer dorsal variant aneurysm (3) arises from the dorsal surface of the ICA distal to the OphArt origin and projects superiorly.

6.1.3 Indications and Contraindications for Surgery

Small (< 1 cm) asymptomatic OphSeg aneurysms, especially those that project into the carotid cave (parasellar variant), are much less likely to rupture compared to lesions more distal in the subarachnoid space.[13] The isolated, incidentally found lesion in an older patient should usually be managed conservatively. Treatment is indicated for virtually all symptomatic aneurysms and for those exceeding 1 cm in size.[9] Patients presenting with visual loss should be treated urgently, ideally with surgery if the patient's risk factors and the experience of the operating team are reasonable. For subarachnoid hemorrhage (SAH) patients, surgery is relatively contraindicated in poor-grade moribund cases with intracranial pressure elevations that cannot be controlled adequately to allow proper brain relaxation and skull base exposures to repair the aneurysm.

6.1.4 Timing of Surgery

Ruptured aneurysms presenting with SAH or epistaxis are treated urgently.[15,16] The surgery can be technically challenging and is usually done during daylight hours. Unruptured but symptomatic aneurysms should also be treated with a sense of urgency, depending on the rapidity of symptom onset and severity of neurologic signs. Elective treatment is reasonable for asymptomatic lesions.[17]

6.1.5 Alternatives to Surgery

Alternative treatments include observation or endovascular treatment. Endovascular techniques include both deconstructive and reconstructive procedures. Reconstruction of the ICA (or repair of the aneurysm) can be done by coiling, stenting, endoluminal flow-diverting devices, or some combination of these.[18] The most appropriate mode of treatment is best determined by a neurosurgeon and an interventional surgeon with expertise in aneurysm treatment, after the patient is stabilized clinically and the radiological workup is complete (which may include a balloon test occlusion of the ICA).[19]

6.1.6 Risks

Specific risks of surgery include death, inadvertent major arterial occlusion causing stroke, incomplete clipping or inability to clip the aneurysm, unilateral blindness and chiasmal distribution visual loss, neurological deficits as a consequence of the surgical exposure, cerebrospinal fluid rhinorrhea, and the systemic complications associated with general anesthesia and a craniotomy. The arteries at risk of occlusion include the ICA, ophthalmic, posterior communicating, and anterior choroidal as well as other perforating arteries.[17,20] ICA occlusion has variable effects ranging from none to hemispheric stroke, brain swelling, and death.

6.2 Preoperative Preparation

Antibiotic prophylaxis, steroids, and mild hypothermia are standard adjuvants, as are a urinary catheter and radial arterial line. Continuous evoked potential and electroencephalographic monitoring are essential in our opinion. Brain relaxation is achieved with wide opening of the sylvian fissure to drain cerebrospinal fluid and with mannitol 0.5 g/kg given 20 minutes before dural opening in patients with SAH. A ventricular drain can be used in patients with SAH. We do not routinely use lumbar spinal drainage. If temporary ICA occlusion is necessary, mild hypertension is induced and intravenous barbiturates are administered to achieve burst suppression on the electroencephalogram.

6.3 Operative Procedure

6.3.1 Positioning and Draping

The patient is placed supine with a shoulder roll underneath the ipsilateral shoulder and the head elevated above the heart to promote venous return. The head is rotated 45 to 60 degrees toward the contralateral side, with the vertex lowered (▶ Fig. 6.4). The maxilla is at the highest point to allow gravitational retraction of the frontal and temporal lobes. Exposure of the cervical ICA may be useful in gaining proximal ICA control. For small and simpler aneurysms, the neck is prepared and draped out without further exposure. For large or complicated aneurysms, especially those presenting with hemorrhage, the cervical ICA should be exposed before the aneurysm is dissected.

6.3.2 Incision and Craniotomy

The skin incision extends from the midline to the zygoma, one fingerbreadth behind the hairline (▶ Fig. 6.4). Care should be taken to spare the major trunk of the superficial temporal artery, as it may be needed for a bypass later in the procedure. The temporalis muscle and fascia are elevated and reflected posteriorly and inferiorly using the Yasargil interfascial technique. A frontotemporal free bone flap is turned up to the edge of the orbital rim to allow an unobstructed view of the orbital roof. An orbital osteotomy can be added to provide additional exposure for larger aneurysms, but generally it is not needed. The lateral sphenoid ridge and the posterior portion of the orbital roof and the lateral orbital wall are removed down to and then above and below the lateral angle of the superior orbital fissure, with a goal of making a flat surface over the orbit connecting the anterior and middle cranial fossa. The lesser wing of the sphenoid is then removed extradurally down to the base of the ACP.

Removal of the ACP, either extradurally or intradurally, is essential during operative treatment of most OphSeg aneurysms. With an extradural removal, the lesser sphenoid ridge medial extension, including the ACP, is hollowed out from within using a high-speed diamond drill until it becomes disconnected at its three points of bony fixation (▶ Fig. 6.5). The tip of the clinoid is then detached from the dura with a microcurette and gently rocked free. Bleeding can be controlled with bone wax and Gelfoam or similar products (Pfizer Inc., New York, NY). The clinoidal space, which contains the clinoidal segment of the ICA, is thus exposed, and this segment can provide proximal ICA control if needed. Extradural clinoidectomy can often be done safely, but should be avoided if a ClinSeg aneurysm is suspected because these lesions can erode into and through the ACP and be injured during its removal.

The authors prefer intradural clinoidectomy (▶ Fig. 6.5). The dura is opened and the sylvian fissure split widely, thus allowing at least part of the aneurysm to be visualized. The falciform ligament is cut by lifting it off the optic nerve with a microhook and cutting it with a no. 11 blade anteriorly until the bony canal is encountered. A 3-cm longitudinal dural incision is then made along the lesser sphenoid wing, beginning at the tip of the ACP and extending laterally and anteriorly beyond the step-off of the extradurally resected lesser sphenoid wing (▶ Fig. 6.5). A second limb of the incision is extended to the sectioned falciform ligament. The dural leaflets are reflected with sutures to expose the remaining portions of the ACP, which is then thinned and removed with a high-speed diamond drill or small rongeur. After disconnecting it from the optic canal and strut, the optic strut is then drilled down to the body of the sphenoid bone to expose the ClinSeg. The optic canal roof and lateral wall are also removed to release the optic nerve, thus allowing the optic nerve to be mobilized medially if necessary. The ICA can be mobilized by circumferential section of the DR, a useful technique for large or giant SupHypArt aneurysms (▶ Fig. 6.6).

6.3.3 Aneurysm Dissection and Clipping

For OphArt aneurysms, the proximal neck begins just distal to the OphArt, but the origin of this branch is often obscured by the ACP (▶ Fig. 6.7). Before aneurysm dissection is begun, the falciform ligament should be sectioned to relax any compression on the superior optic nerve surface. The distal neck is typically free of perforators, so a gently curved or side-angled clip can usually be applied parallel to the course of the ICA, taking care to spare the OphArt or any perforators arising from its superior surface. Calcification along the anterior aspect of the aneurysm may prevent closure of the clip blades. In such

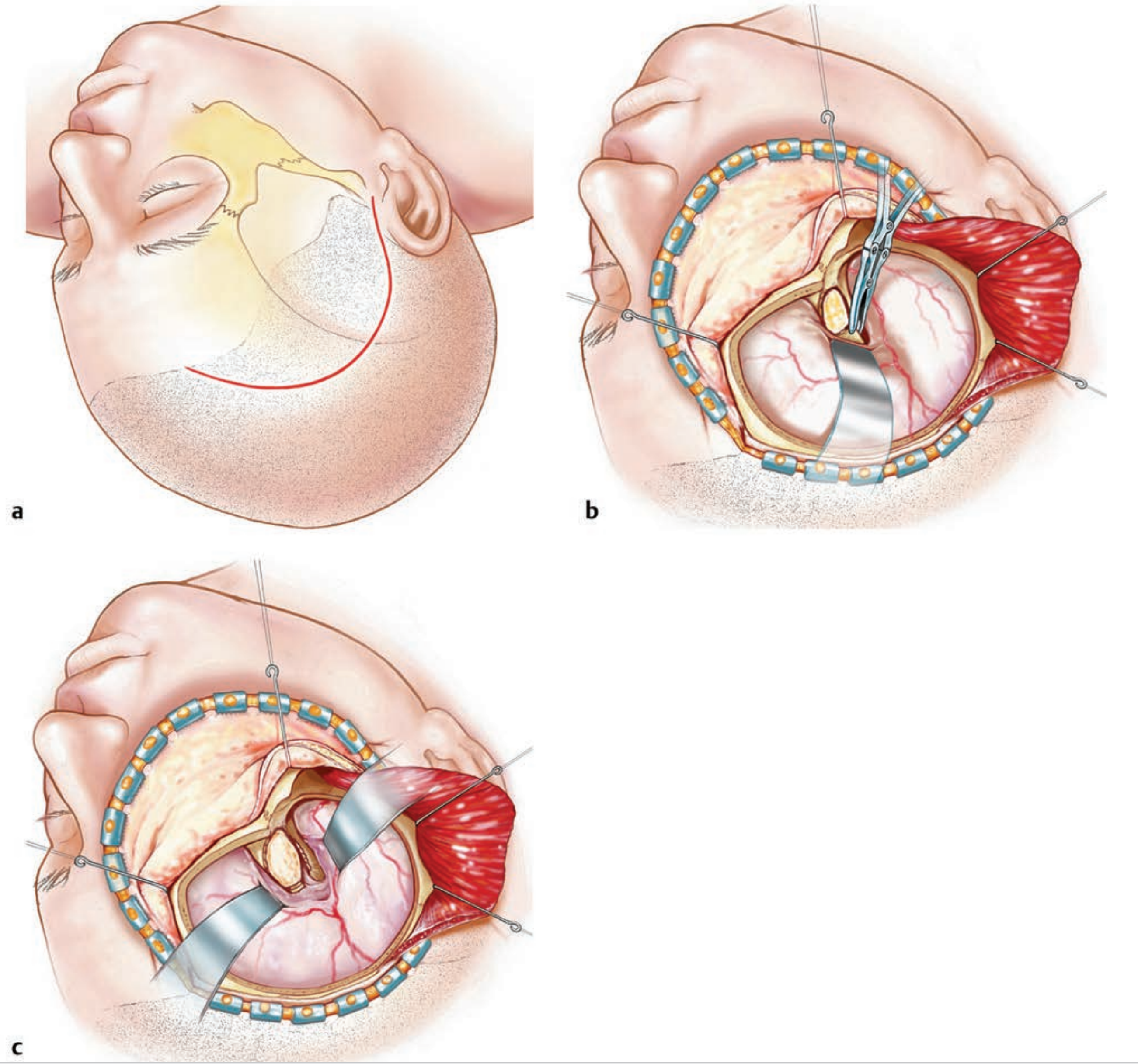

Fig. 6.4 Positioning, skin incision, and extradural bone removal. **(a)** Scalp incision is from midline to zygoma. **(b)** The sphenoid ridge is flattened and the posterior orbital roof and lateral orbital wall are removed. **(c)** Extradural bone removal can be extended to include the anterior clinoid process.

circumstances, fenestrated clips can be used to encircle the calcified portion of the aneurysm. Clipping too close to the aneurysm neck must be avoided because the internal lumen of the ICA can be compromised by ICA atherosclerosis. Once the aneurysm is clipped and ICA patency confirmed, the aneurysm dome can be opened and its contents evacuated to decompress the optic apparatus.

SupHypArt aneurysms typically project medially away from the lateral surface of the surgically exposed ICA. As they enlarge, their walls become adherent to the dura of the diaphragma sella and lateral cavernous sinus wall. Although the angiogram may suggest otherwise, they do not project into the cavernous sinus, and the walls of the aneurysm can be separated from the venous walls. Initial exposure of the medial aneurysm neck is similar to that for an OphArt aneurysm and involves opening of the opticocarotid cistern, falciform ligament, and optic canal. The ClinSeg should be exposed and dissection carried proximal to the DR to expose the proximal aneurysm neck and origin of the SupHypArt within the carotid cave. The hypophyseal stalk may be adherent to the posterior and medial surface of the aneurysm, and the PComArt or its thalamoperforating branches are often draped over the distal end of the aneurysm. These structures must be identified, dissected free, and preserved. The aneurysm is usually best clipped with fenestrated clips to encircle the ICA with the clip blades running at right angles parallel to the ICA (▶ Fig. 6.7). When clipping calcified aneurysms and to avoid problematic SupHypArt compromise, the proximal portion of the aneurysm

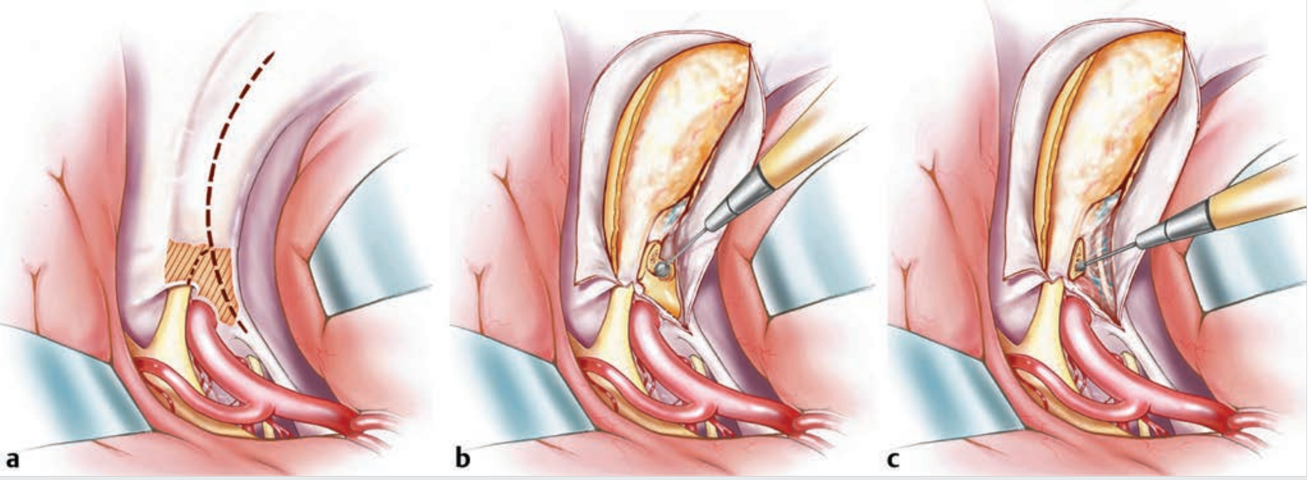

Fig. 6.5 Intradural anterior clinoidectomy. **(a)** A longitudinal dural incision is made along the sphenoid ridge to the tip of the anterior clinoid process. A transverse incision is made toward the falciform ligament. **(b)** The anterior clinoid process and the roof of the optic canal are removed with rongeurs and a high-speed diamond drill. **(c)** After removal of the anterior clinoid process, the optic strut is drilled down to its basal attachment to the sphenoid bone.

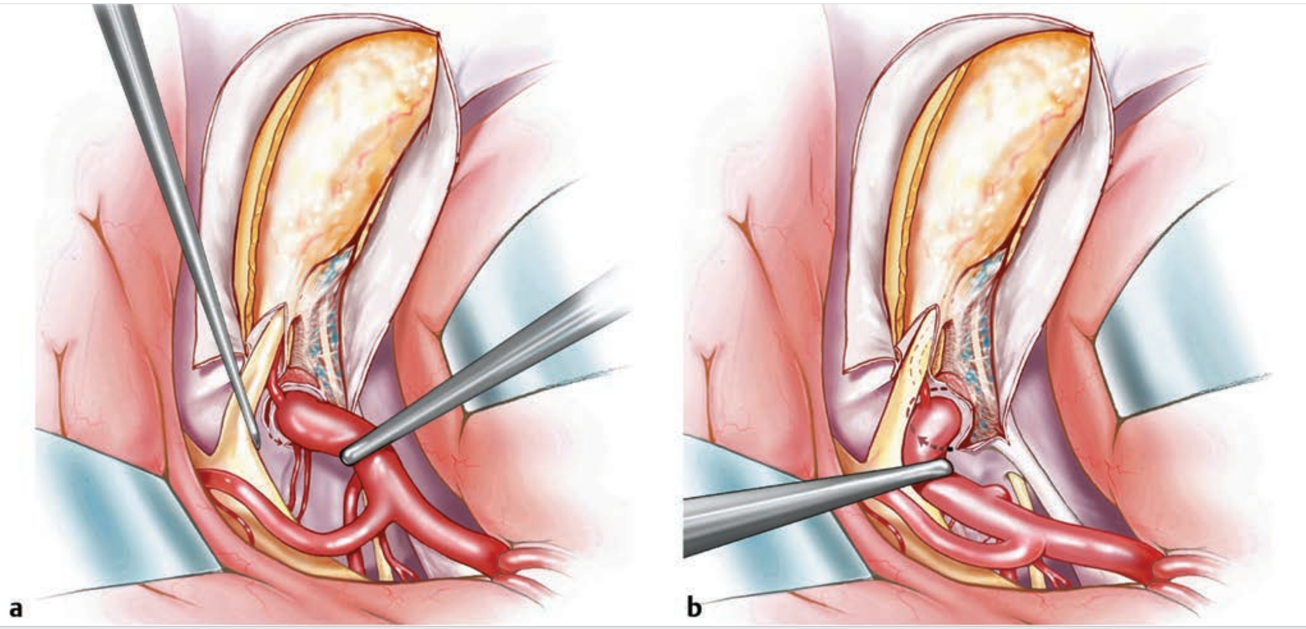

Fig. 6.6 Sectioning of the dural ring along the **(a)** medial and **(b)** lateral aspect of the internal carotid artery allows for mobilization of the artery.

can be obliterated with perpendicularly placed short-bladed fenestrated straight clips stacked to harness their additional closing pressure (▶ Fig. 6.7). Circumferential section of the DR allows the clip blades to pass unencumbered across the proximal neck without kinking of the SupHypArt origins.

Dorsal carotid wall blister aneurysms are extremely fragile and can rupture easily during clip placement. The clip should be applied only after trapping of the affected segment. The clip blades should ideally be placed parallel to the long axis of the ICA to avoid any twisting or torque on the fragile parent vessel. A sling of fascia or Gore-Tex can be used to “wrap” the segment, with the clip bringing the two edges together, completely encircling and supporting the diseased segment.

Temporary clipping of the cervical ICA can be used to soften large aneurysms sufficiently to allow for clip reconstruction. An intraoperative angiogram is highly recommended to ascertain parent vessel patency and complete aneurysm obliteration. Indocyanine green allows visualization of the perforator patency and can substitute for angiography in some instances.

6.3.4 Closure

Any communication of the ACP with the sphenoid sinus must be identified and sealed with packing (Gelfoam and/or muscle and methylmethacrylate) to prevent cerebrospinal fluid leakage. Opening of the craniotomy into the frontal sinus should be

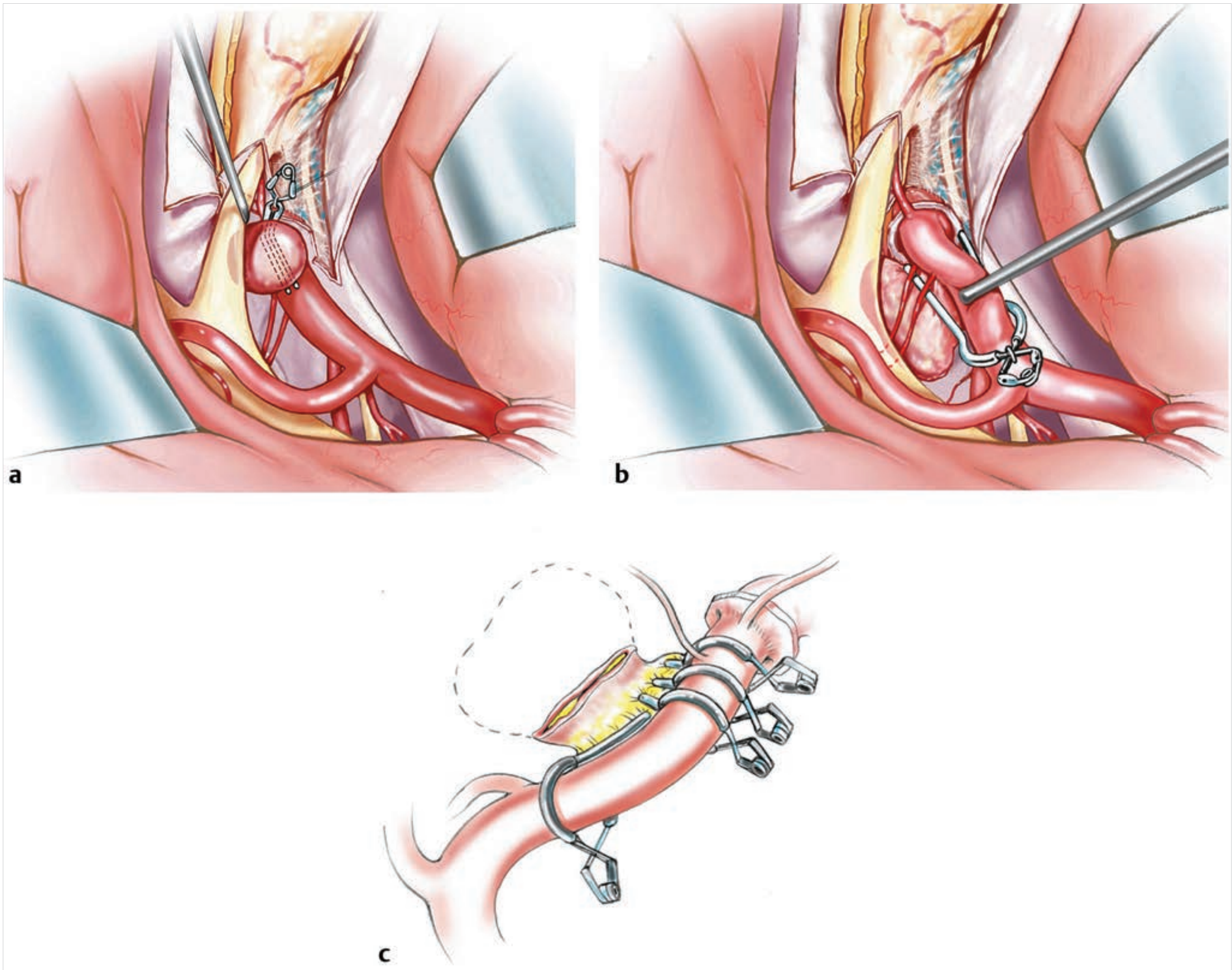

Fig. 6.7 Clipping techniques. **(a)** An OphArt aneurysm is clipped using a side-angled or gentle curved clip. The clip blades are placed between the OphArt and the aneurysm and applied parallel to the internal carotid artery (ICA). **(b)** A SupHypArt aneurysm is clipped with a fenestrated right-angled clip to reconstruct the ICA lumen. The heel of the clip must spare the PComArt, while the tip of the clip extends up to or proximal to the dural ring. **(c)** A calcified SupHypArt aneurysm is clipped using a series of fenestrated clips.

repaired. Replacement of the bone flap and the rest of the closure follow that of a standard craniotomy.

6.4 Postoperative Management Including Complications

The two main complications are visual loss and ischemia in the territory of the ipsilateral ICA. Visual deterioration may occur if the optic nerve, already distorted medially and superiorly by the underlying aneurysm, is further manipulated against the falciform ligament. This risk may be minimized by sectioning the falciform ligament and unroofing the optic canal before aneurysm dissection. Occlusion of the OphArt or superior hypophyseal perforators may impair blood supply of the optic nerve and chiasm and cause visual loss. As with aneurysms in other locations, the clip should be carefully inspected following final placement to verify that no perforating arteries have inadvertently been clipped.

Transient or fixed postoperative hemibody deficits may indicate ICA compromise and occur with higher frequency in patients with calcified or partially thrombosed aneurysms with atherosclerosis in the ICA wall. Intraoperative angiography is essential when ICA patency is questionable at surgery or when distal embolization is considered possible. Patients awakening with deficits need emergent CT scanning and possibly angiography, particularly if intraoperative studies were not done.

Postoperative diplopia may be due to either an abducens or oculomotor nerve paresis. When the DR is opened, these nerves lie in a relatively superficial position within the wall of the clinoidal space. They may be disturbed within the cavernous sinus either by clinoid removal or by the clip blades as they are advanced proximally beyond the aneurysm neck. These cranial nerve deficits are usually transient.

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7 Supraclinoid Internal Carotid Artery Aneurysms

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Abstract

The supraclinoid internal carotid artery constitutes a frequent site for aneurysm development. The aneurysms along the internal carotid artery (ICA) are associated with the ophthalmic artery, posterior communicating artery, anterior choroidal artery, and ICA bifurcation. Microsurgery plays an essential role in the treatment of these lesions. Preoperative planning using three-dimensional computed tomography (3D-CT) or catheter angiography is key in order to anticipate the surgeon's operative view. We then use a lateral supraorbital craniotomy to approach many of these aneurysms. Once the aneurysm is felt to be secure, indocyanine green videoangiography and Doppler ultrasound are used to confirm aneurysm exclusion and parent and branch artery patency.

Keywords: cerebral aneurysm, internal carotid artery, microsurgery

7.1 Indications, Contraindications, and Alternatives

The internal carotid artery (ICA) from the distal dural ring to the ICA bifurcation is a frequent site of aneurysms development, and includes the ophthalmic artery, posterior communicating artery, anterior choroidal artery, and ICA bifurcation. Intracranial aneurysms of the ICA and its branches constituted about 25% of all intracranial aneurysms.

Intracranial aneurysms may be detected incidentally in an asymptomatic patient, in which case the first decision is whether to observe or treat the aneurysm. Aneurysms may present symptoms and signs caused by the aneurysm mass, by embolism or hemorrhage. Whether to repair an aneurysm depends on its clinical presentation, whether the aneurysm is asymptomatic or symptomatic, and unruptured or ruptured. In addition, patient-specific risk factors for rupture/rerupture, affinity for follow-up, chances of successful treatment overall and by clipping or coiling, complications of these treatments, and characteristics of the aneurysm (location, size, morphology, calcification, intra-aneurysmal thrombus, presence of other aneurysms) all have to be entered into the risk–benefit analysis in order to make educated management decisions.[1] Even after the decision to treat has been made, there are endovascular and open microsurgical options. Choosing the appropriate treatment strategy is discussed elsewhere (Chapter 2) and is based on varying levels of medical evidence. For ruptured aneurysms, endovascular coiling and neurosurgical clipping are the main options, and the decision as to the method of aneurysm repair should be based on the characteristics of the patient (age, other illnesses) and their clinical condition (neurological grade) as well as on the characteristics of the aneurysm (location, size, presence of thrombus or calcification, associated aneurysms, space-occupying intracerebral hematoma), to mention a few.

Preoperative planning for the microsurgical treatment of intracranial aneurysms includes three-dimensional computed tomography (3D-CT) and/or 3D catheter angiography. 3D-CT angiography provides valuable information regarding the bony landmarks and their relationship with the aneurysm for proper surgical orientation.[2]

Contraindications to treatment of ICA aneurysms are rarely absolute. Basically, the managing physician has to consider the best available evidence about the probabilities of what will happen if: (1) the aneurysm is not treated and the outcome is related to the natural history that has been reported in patients with a similar aneurysm compared to (2) the aneurysm was treated, which has to combine the risks of treatment with the efficacy of the treatment or how much (and the scientific evidence for which) the treatment reduces the risk of (1).

7.2 Lateral Supraorbital Approach

Hernesniemi has been using the lateral supraorbital approach for more than 20 years.[3] It is based on a pterional craniotomy but uses a smaller skin incision, less muscle dissection, and less bone removal while achieving adequate access to the majority of anterior circulation aneurysms and some posterior circulation aneurysms as well.

7.2.1 Positioning

The patient is positioned supine with the head and shoulders elevated above the cardiac level and head rotated 15 to 30 degrees to the contralateral side. The head is fixed with neck slightly flexed and tilted laterally in a Sugita frame.

7.2.2 Craniotomy

An 8- to 10-cm oblique frontotemporal skin incision is made just behind the hairline (▶ Fig. 7.1a). A myocutaneous flap is raised with only the needed anterior portion of the temporalis being dissected. This minimizes postoperative muscle atrophy and risk of injury to the frontalis branch of the facial nerve.

The flap is retracted anteriorly with spring hooks until the zygomatic process of the frontal bone is exposed (▶ Fig. 7.1b). One burr hole is made at the posterior aspect of the exposed superior temporal line. A bone flap (approximately 4 cm × 4 cm) is made using a side-cutting drill (▶ Fig. 7.1c). The most inferior aspect of the craniotomy should expose a portion of the temporal lobe. Once the flap is lifted, the craniotomy is refined using a diamond burr to provide greater access while simultaneously using the generated heat to coagulate bleeding bone. In particular, the vertical bone and lateral sphenoid ridge are drilled to provide better access to the sylvian cistern. The dura is opened with a posteriorly arcing incision based on the fronto-orbital bone and retracted with multiple tack-up sutures to prevent obstruction of the surgical field and to tamponade epidural bleeding (▶ Fig. 7.1d). At this point, the operative microscope is brought into the field.

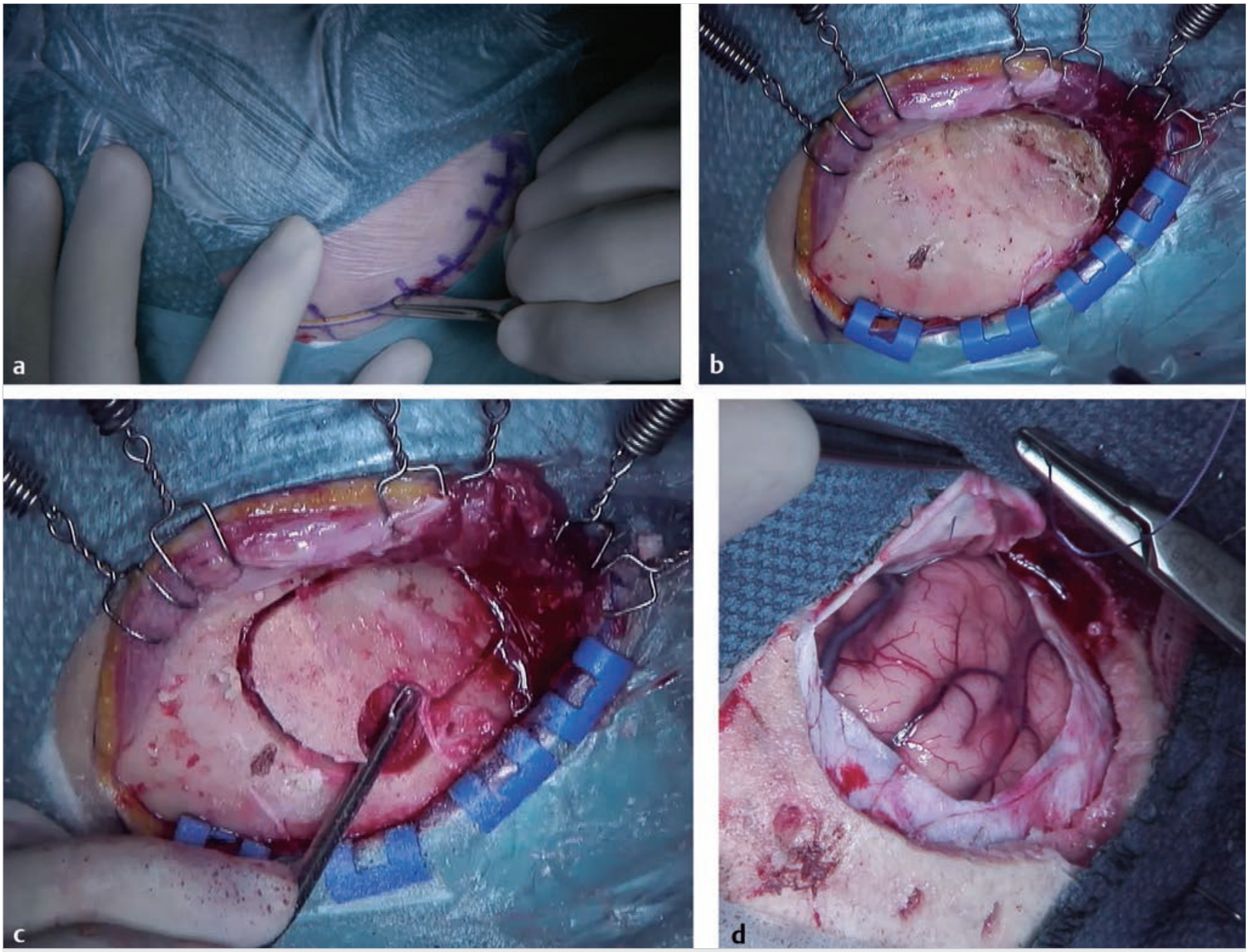

Fig. 7.1 **(a)** A curvilinear frontotemporal skin incision is made just behind the hairline. **(b)** The myocutaneous flap is retracted anteriorly as a one-block layer flap. The key points of the approach are the zygomatic process of the frontal bone, frontozygomatic suture, superior temporal line, and the projection of the sylvian fissure over the bone. **(c)** A single burr hole is placed over the superior temporal line. The side-cutting drill is used to create a craniotomy of approximately 4 × 4 cm. **(d)** The dura is opened with a posteriorly arcing incision based on the fronto-orbital bone and retracted with multiple tack-up sutures.

7.3 Posterior Communicating Artery Aneurysms

In 1938, Dandy reported neurosurgical clipping of a posterior communicating artery aneurysm, probably describing the first such attempt at direct intracranial aneurysm clipping.[4] The patient presented with third cranial nerve palsy and was diagnosed with a posterior communicating aneurysm. Aneurysms at this location represent 12 to 25% of all intracranial aneurysms.[5,6,7]About 20% present with third cranial nerve palsy that may or may not improve with surgery.[8,9] Third nerve palsy tends to recover more completely if the aneurysm is repaired sooner after onset of the palsy and also sooner (84 vs. 137 days) and more completely (99 vs. 68%) with microsurgery compared to endovascular treatment.[9]

Posterior communicating artery aneurysms are suitable for microsurgical clipping due to their origin off the communicating segment of the ICA. This location of the aneurysms allows the surgeon to achieve proximal and distal control of the parent artery to isolate the aneurysm. These aneurysms are also sufficiently proximal so that they do not always require opening of the sylvian fissure, but if more than minimal frontal lobe retraction is needed, the sylvian fissure should be split proximally. It is important that the sylvian fissure is oriented vertically during the positioning. Sharp arachnoid dissection permits identification of the optic nerve and ICA. Opening of the chiasmatic cistern at this time relaxes the brain. The ICA is dissected on its medial and lateral walls while moving along the vessel distally (▶ Fig. 7.2). The optico-carotid triangle should be completely opened to expose the posterior communicating artery, which arises from the posterolateral aspect and runs medially and posteriorly to join the posterior cerebral artery.

The posterior communicating artery often is difficult to visualize proximally as it turns to run medially behind the ICA just after it branches off of the ICA. This origin can be found by tracing the portion in the optico-carotid triangle more proximally or by tracing the anterior dome of the aneurysm to the point where its neck meets the inferior aspect of the

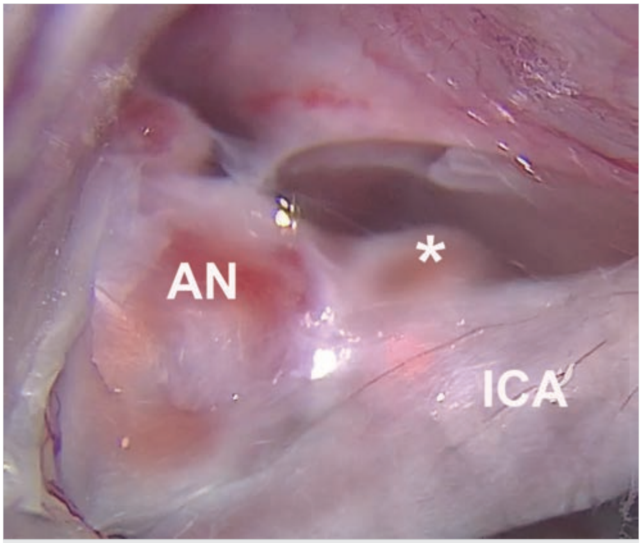

Fig. 7.2 A left small unruptured posterior communicating artery aneurysm exposed through a lateral supraorbital approach. The dissection starts over the medial and lateral walls of the ICA, moving from proximal to distal to secure a place for temporary clip placement. *, posterior communicating artery, AN, aneurysm, ICA, internal carotid artery.

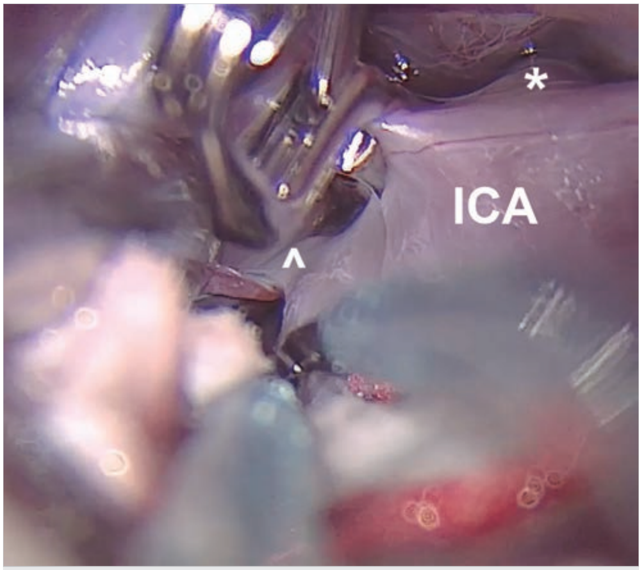

Fig. 7.3 After final clipping of a left unruptured posterior communicating artery aneurysm. The following step is to identify the posterior communicating artery and anterior choroidal artery. *, posterior communicating artery; ^, anterior choroidal artery; ICA, internal carotid artery.

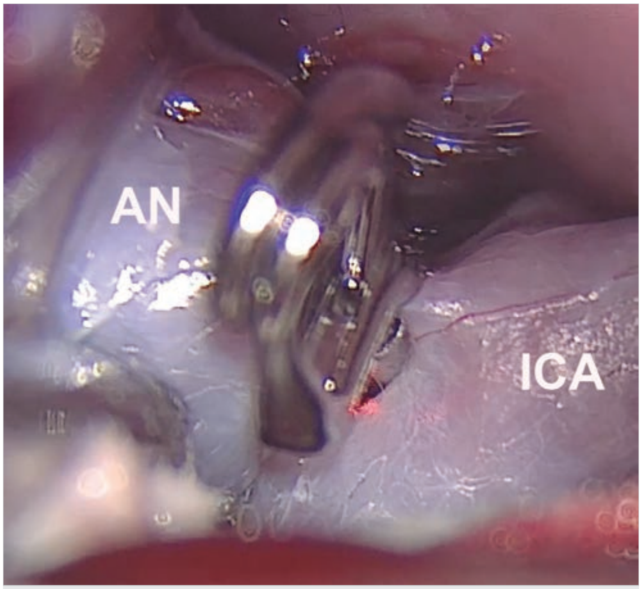

Fig. 7.4 Final clipping with a straight clip. AN, aneurysm; ICA, internal carotid artery.

ICA.[10] It is important to also identify the origin of the anterior choroidal artery so as not to incorporate it into the clip (▶ Fig. 7.3 and ▶ Fig. 7.4). The neck of the aneurysm may be partially obscured by the anterior clinoid process and sometimes requires a clinoidectomy for complete exposure.

Posterior communicating artery aneurysms are typically oriented in a lateral direction and can be intimately related to the third cranial nerve, the tentorium, and the uncus. The temporal lobe should not be retracted if the aneurysm is projecting toward it, as it may be adherent if sitting above the tentorium and this could cause intraoperative rupture. If the aneurysm is adherent to the third cranial nerve, it should be dissected free carefully to avoid iatrogenic third nerve palsy due to traction (**Video 7.1**).

Giant and large posterior communicating artery aneurysms have an incidence of 4 to 7% among giant intracranial aneurysms.[11,12] Due to the complexity of these lesions, each particular case should be planned carefully. Treatment options include direct surgical clipping with parent artery preservation, aneurysmorrhaphy, carotid ligation, revascularization procedures, and endovascular methods (coiling, stent placement, flow-diverters).

7.4 Anterior Choroidal Artery Aneurysms

The majority of anterior choroidal arteries originate directly from the ICA trunk just distal to the posterior communicating artery. An origin from the posterior communicating artery or the middle cerebral artery has been described.[13,14] The anterior choroidal artery usually arises closer to the origin of the posterior communicating artery than to the ICA bifurcation. Moreover, the anterior choroidal artery may arise as a single trunk or as multiple vessels from the posterolateral wall of the ICA.

The treatment of anterior choroidal artery aneurysms is difficult due to the presence of several perforators, its important vascular distribution, and anatomical variations. These lesions account for 2 to 5% of all intracranial aneurysms.[15,16] These aneurysms can be classified according to their dome orientation and origin of the anterior choroidal artery aneurysm: (1) “anterolateral” meaning in front of and obscuring the origin of the anterior choroidal artery; (2) “superolateral” meaning distal to the origin of the anterior choroidal artery; (3) “posterolateral” meaning behind the origin of the anterior choroidal artery; (4) between duplicated anterior choroidal arteries or perforating branches of the anterior choroidal artery.[17] This classification provides important details for surgical planning and positioning of the patient's head. Other important considerations when planning surgery should include possible calcifications of the supraclinoid ICA, anatomic variations of both the anterior choroidal and the posterior communicating arteries, ventricular hemorrhage, and the relationship of the aneurysm with the anterior or posterior clinoid processes.

7.4.1 Microsurgery

Posteriorly projecting anterior choroidal artery aneurysms require the head to be tilted more to the contralateral side than laterally projecting aneurysms. Overrotation should be avoided, because the temporal lobe may obstruct the sylvian fissure making the focused opening challenging and could also block the surgeon's view of the ICA.[18]

Opening of the optico-carotid cistern creates enough working space during the dissection phase. If more room is required, the membrane of Liliequist can be opened to release additional cerebrospinal fluid. An anterior clinoidectomy is sometimes necessary to provide extra space for aneurysm visualization and temporary clip placement. The dissection continues along the anterior, medial, and lateral walls of the ICA toward the ICA bifurcation. The use of sharp dissection prevents stretching of the ICA and tearing the aneurysm dome. Once the proximal part of the M1 segment is exposed, the dissection should continue along the anteromedial wall of the ICA until identification of the ICA bifurcation complex and the proximal A1 and M1 segments. After careful sharp dissection of the arachnoid bands under the frontal lobe, a retractor placed subfrontally may further facilitate the exposure. An important step is to allocate a proper site (free of perforators) for temporary clip placement on the distal ICA trunk when possible or the proximal M1 and A1 segments. In all the steps, retraction of the temporal lobe should be avoided if possible to prevent tearing of the aneurysm (**Video 7.2**).

The Helsinki clipping dogma is to first apply a pilot clip over the anterior choroidal artery aneurysm neck. The pilot clip is then exchanged for a smaller and lighter final clip for complete occlusion of the aneurysm neck. For small aneurysms, the double-clipping technique is used. Temporarily clips are removed carefully in counter order.

7.5 ICA Bifurcation Aneurysms

Aneurysms of the ICA bifurcation are located at the division of the ICA to the anterior and the middle cerebral arteries. The proximal portions of the aforementioned arteries can be involved in aneurysm formation. ICA bifurcation aneurysms are uncommon, with an incidence of 2 to 9% of all intracranial aneurysms.[2,19,20,21] However, they seem to affect younger patients with a higher frequency; 28% of all intracranial aneurysms in patients less than 20 years of age are located at the ICA bifurcation.[22,23,24] About 50% of all ruptured ICA bifurcation aneurysms are smaller than 7 mm.

7.5.1 Microsurgery

The direct microsurgical treatment of ICA bifurcation aneurysms is challenging due to the high location of the ICA bifurcation in relation to the skull base, attached brain parenchyma, and perforating branches to the anterior perforated substance. Therefore, the head should be extended as much as safely permissible during positioning to provide optimal visualization of the ICA bifurcation with minimal brain retraction.[2] ICA bifurcation aneurysms are classified based on orientation of the aneurysm dome as anteriorly, superiorly, or posteriorly projecting aneurysms.[19,25,26]

If excessive temporary occlusion times are anticipated or there is a possibility that trapping may be required, a preoperative digital subtraction angiography with balloon test occlusion can provide valuable information about collateral circulation and the patency of the anterior communicating artery complex.[27,28]

In cases of ruptured ICA bifurcations aneurysms with an associated intracerebral hematoma, the clot can be partially evacuated from a small cortical incision that avoids and preserves Broca's area. Opening of the suprasellar and chiasmatic cisterns as well as the lamina terminalis through the subfrontal approach provides additional space and brain relaxation.[29]

In our experience, the best surgical route to the ICA bifurcation is to open the carotid cistern and the proximal sylvian cistern to divide the proximal "horizontal" portion of the sylvian fissure. These maneuvers allow direct visual control of the perforating branches, the proximal group of perforators arising from the precommunicating segment of the anterior cerebral artery, the medial lenticulostriate arteries from the first segment of the middle cerebral artery, and the recurrent artery of Heubner (▶ Fig. 7.5).[2,30] A wider sylvian fissure dissection to the level of the middle cerebral artery bifurcation may be required for superiorly or posteriorly projecting aneurysms.

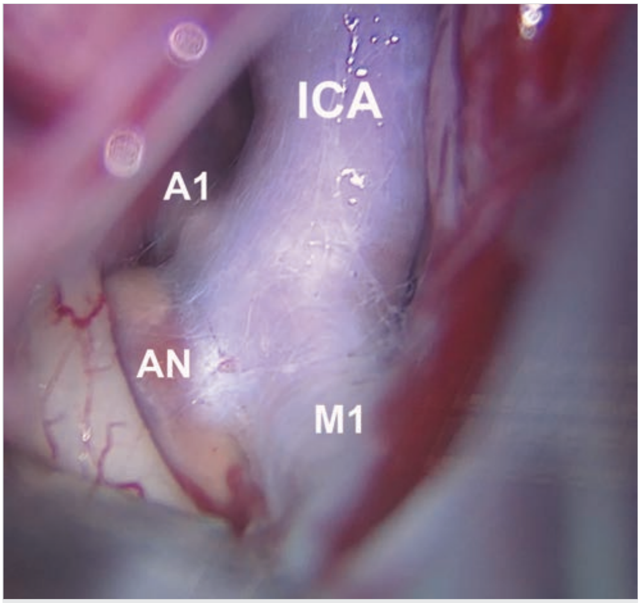

Fig. 7.5 A small right unruptured ICA bifurcation aneurysm approached through a right lateral supraorbital approach. Subfrontal dissection and partial opening of the proximal segment of the sylvian fissure was performed to expose the aneurysm and the proximal segments of the anterior and middle cerebral arteries. A1, proximal segment anterior cerebral artery; AN, aneurysm; ICA, internal carotid artery; M1, proximal segment middle cerebral artery.

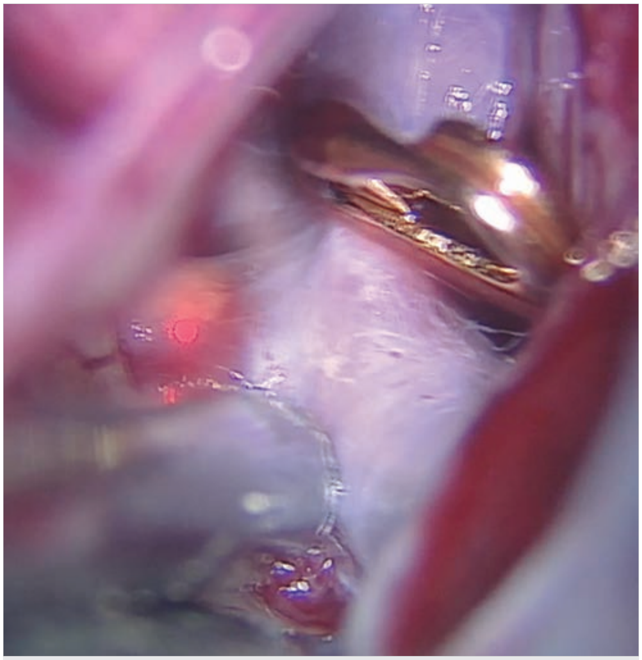

Fig. 7.6 A temporary clip was placed proximal to the ICA bifurcation.

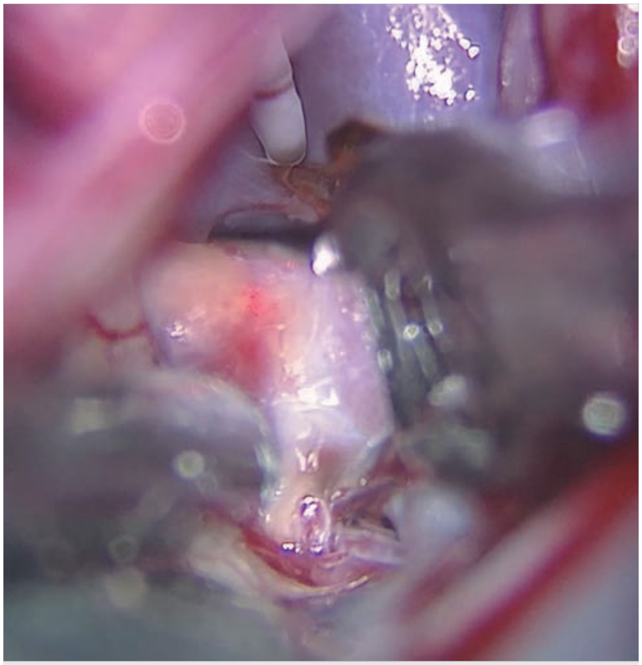

Fig. 7.7 A straight clip was used to occlude the neck of the aneurysm while the temporary clip was in placed.

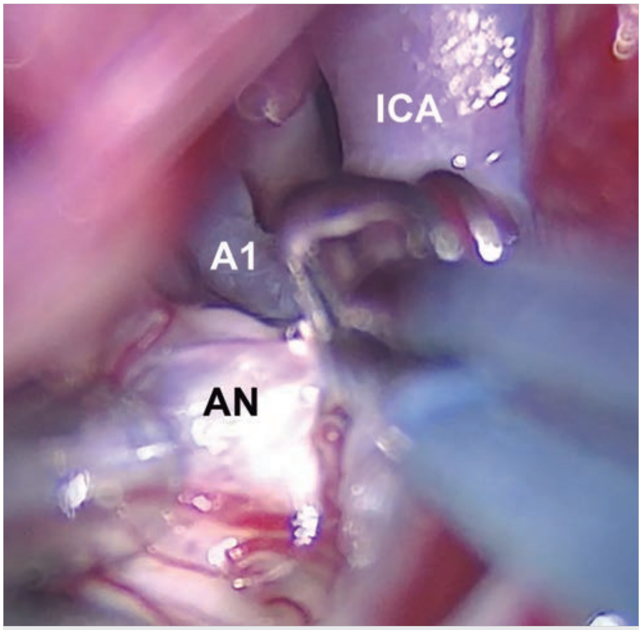

Fig. 7.8 Once the final clip was placed, the temporary clips are removed and the patency of the parent artery and small perforators is confirmed. A1, proximal segment anterior cerebral artery; AN, aneurysm; ICA, internal carotid artery.

Frontal lobe retraction should be performed carefully to avoid intraoperative rupture, particularly in superiorly or anteriorly projecting aneurysms that are often attached to the overlying frontal lobe.

Small perforators arising from the ICA should be identified before placing a proximal temporary clip between the ICA bifurcation and anterior choroidal artery (▶ Fig. 7.6). If there is no space, temporary clips can be placed on the proximal middle and anterior cerebral arteries. ICA bifurcation aneurysms (**Video 7.3**), in general, are not often large or giant in size, making their clipping feasible using straight clips (▶ Fig. 7.7 and ▶ Fig. 7.8). Special care should be taken with posteriorly projecting ICA bifurcation aneurysms because of the small perforators arising from the posterior aspect of the bifurcation. These aneurysms may also be adherent to the anterior choroidal artery.[17,31,32]

If the ICA bifurcation aneurysm is complex and involves the proximal portions of the anterior or middle cerebral arteries, different strategies are required if direct clipping is not possible. Revascularization procedures have to be considered when trapping or proximal occlusion of the ICA bifurcation is necessary.

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8 Anterior Communicating Artery Aneurysms

E. Francois Aldrich, Elizabeth Julianna Le, and J. Marc Simard

Abstract

The anterior communicating artery (ACoA) complex is the most common site of intracranial aneurysms, representing approximately 30% of aneurysms associated with subarachnoid hemorrhage.[1] The heterogeneity in the anatomy of the ACoA complex, variable origin and structure of the aneurysms, dome projections and their relationship to the skull base and optic nerves, and the numerous critical perforating arteries originating in the area can result in challenging cases that should be individually studied and understood prior to treatment. Though endovascular treatment is increasingly used for these aneurysms, given their inherent complexity, surgical obliteration will remain an important treatment modality.[2]

Keywords: anterior communicating artery, cerebral aneurysm, subarachnoid hemorrhage

8.1 Anatomic Considerations

The region of the anterior communicating artery (ACoA) has an extremely variable normal anatomy (▶ Fig. 8.1). The complex relationships between the blood vessels, aneurysm, and other surrounding structures make ACoA aneurysms one of the most complex to treat surgically. The following factors should be thoroughly analyzed on high-quality computed tomography angiography with three-dimensional reconstructions and/or digital subtraction angiography for each patient:

- The configuration of the aneurysm sac and the direction of projection.
- The anatomy of the neck of the aneurysm.
- The relationship of the aneurysm to the ipsilateral and contralateral pre- (A1) and postcommunicating (A2) segments of the anterior cerebral arteries (ACAs).
- The side of the dominant A1 segment.
- Anatomic variations.

The caliber of the A1 segment is asymmetric in 85% of patients with ACoA aneurysms, with 10% having a hypoplastic A1 segment.[3] This asymmetry almost invariably correlates with the aneurysm orientation as the base of the aneurysm typically emerges on the side of the larger A1 segment with the dome pointing toward the side of the hypoplastic segment. Thus, the aneurysm sac will possess a distinct orientation, which has important surgical implications and can be classified based on its relationship to the planum sphenoidale:

- Inferior (toward the planum).
- Anterior (toward the nose, parallel to the planum).
- Superior (toward the vertex, perpendicular to the planum).

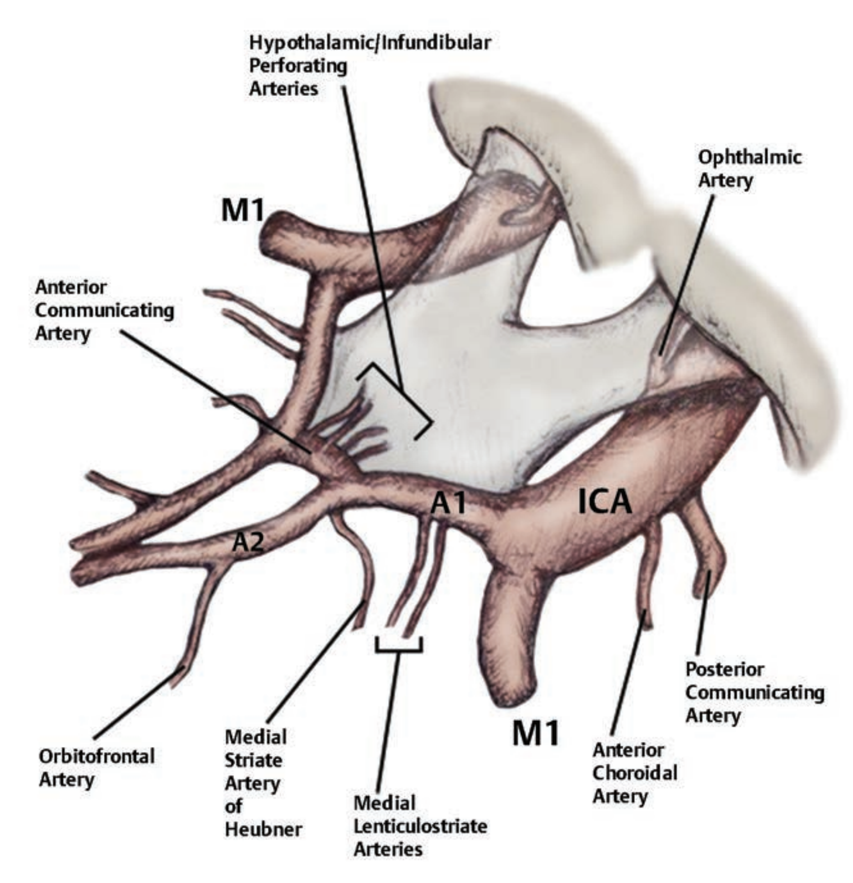
Hypothalamic/Infundibular Perforating Arteries
Ophthalmic Artery
M1
Anterior Communicating Artery
A1
ICA
A2
Posterior Communicating Artery
M1
Orbitofrontal Artery
Medial Striate Artery of Heubner
Medial Lenticulostriate Arteries
Anterior Choroidal Artery

Fig. 8.1 Anatomy of anterior communicating artery region.

- Posterior (toward the occiput, parallel to the planum).
- Circumferential (ectasia of the ACoA).

Combinations of these projections are frequent, particularly in large aneurysms.

The ACoA may also demonstrate anomalous anatomy, with approximately 25% ranging from a network of multiple bridges to duplication or triplication of the artery. Furthermore, the A2 displays variability. In the majority (90%) of cases, there are two A2 arteries, but three A2 arteries can be found in 9% and a single (azygos) A2 artery can be seen in 1%. Another significant area of divergence in anatomy occurs with the recurrent artery of Heubner (medial striate artery), the largest and most important of the striate perforators in the ACA–ACoA complex. This artery originates from the A2 segment in 78% of cases, from the A1 segment in 14% of cases, and at the level of the ACoA in 8% of cases, curving sharply back on itself to course parallel to the A1 segment.[4] The size and position of this artery correspond to other components of the ACoA complex. For instance, in cases of small A1 arteries, the recurrent artery of Heubner may also be small or atretic with possible replacement by large medial perforating arteries that originate from the midportion of the A1 segment and travel more laterally than the typical recurrent artery of Heubner.

Another critical anatomical consideration is the relative parallel or perpendicular orientation of the ACoA to the planum sphenoidale, which influences the visualization of the contralateral A1 and A2 arteries and the neck of the aneurysm, as well as the aneurysm clip selection and final placement (▶ Fig. 8.2 and ▶ Fig. 8.3a,b).

8.2 Surgical Approaches

Once the aneurysm configuration and anatomic considerations have been thoroughly analyzed, the type and side of approach can be determined. ACoA aneurysms can be approached from an anterior (interhemispheric), anterolateral (subfrontal), lateral (pterional), extended skull base, or combination of these approaches. In our practice, the interhemispheric approach is not routinely used unless the aneurysm is superiorly projecting, located at least 15 mm above the anterior clinoid process, and the remaining anatomy is otherwise favorable. Advantages of the interhemispheric approach include less need for frontal lobe retraction and easier visualization of the ACoA complex and adjacent vessels. However, the main disadvantage is that the aneurysm sac is frequently encountered prior to dissection of the neck, particularly in anteriorly projecting and large aneurysms. In such cases, adequate aneurysm neck exposure and satisfactory proximal control can be difficult to obtain. Additionally, both frontal lobes are subject to potential injury, the operative field increases in depth, the olfactory tracts are at increased risk of injury, and entry into the frontal sinus elevates the risk of infection and cerebrospinal fluid leakage. Although the subfrontal approach provides the most direct course to the ACoA complex, the disadvantages are similar to those of the interhemispheric approach, primarily encountering the aneurysm dome prior to obtaining proximal control. The pterional approach with emphasis on a larger frontal exposure is the most frequently advocated approach for ACoA aneurysms and used in virtually all cases in our practice. We reserve the use of extended skull base approaches, such as supraorbital, transorbital, and orbitocranial zygomatic craniotomies, for specific complicated or giant aneurysms.

Once an approach has been selected, the side of operation must be determined. It is our practice to virtually always approach an aneurysm from the side of the dominant A1, if one is present, as it allows greater visualization and easier dissection of the aneurysm neck without disturbance of the dome since the aneurysm sac typically projects away from the dominant A1. Additionally, earlier access to the dominant A1 can be obtained for proximal control. However, it should be emphasized that temporary clipping of only the dominant A1 without temporary clipping of the contralateral A1 virtually never results in complete vascular control. Conversely, approaching the ACoA aneurysm from the side of the nondominant A1 may be considered in the presence of other aneurysms of the anterior circulation that the surgeon feels compelled to treat concurrently, as well as in cases of large intraparenchymal hemorrhages or unilateral gyrus rectus hematomas requiring evacuation or to preserve the uninjured parenchyma. However, if the surgical factors favoring each side are otherwise equal, the aneurysm should be approached from the patient's nondominant side.

8.3 Operative Procedure

8.3.1 Exposure

A pterional craniotomy is performed in the standard fashion with specific considerations made for ACoA aneurysms. Horizontal head rotation is no greater than 45 degrees in the contralateral direction, which is less than for other anterior circulation aneurysms. The frontal portion of the bone flap is larger than for other anterior circulation aneurysms and extends laterally to the midpupillary line, which is approximately 3 cm from the zygomatic orbitofrontal junction. Placement of frontal burr holes below the hairline is avoided for cosmesis. In order to minimize retraction on the frontal lobe, striving to be as low as possible on the anterior fossa floor is essential in this extended frontal approach. Although opening of the frontal sinus increases the risk of infection and cerebrospinal fluid leakage, an extensive view of the anterior fossa floor is crucial and takes precedence. The temporal portion of the craniotomy needs only to be large enough to expose the proximal aspect of the sylvian fissure.

8.3.2 Microdissection

After the dura is opened, the first step in microdissection is identification of the optic nerve, carotid artery, and ipsilateral A1 artery (▶ Fig. 8.2a). A frontal retractor is placed on the orbital surface of the frontal lobe immediately anterior to the sylvian fissure. Advancing the retractor blade beyond the olfactory nerve to the gyrus rectus and applying slight frontal lobe retraction will identify the ipsilateral optic nerve. The opticocarotid cistern is opened exposing the carotid artery. A retractor is then placed over the temporal lobe. Slight tension will expose the proximal sylvian fissure, allowing division of the most medial and inferior region. We routinely strive to preserve the temporal bridging veins during this dissection.

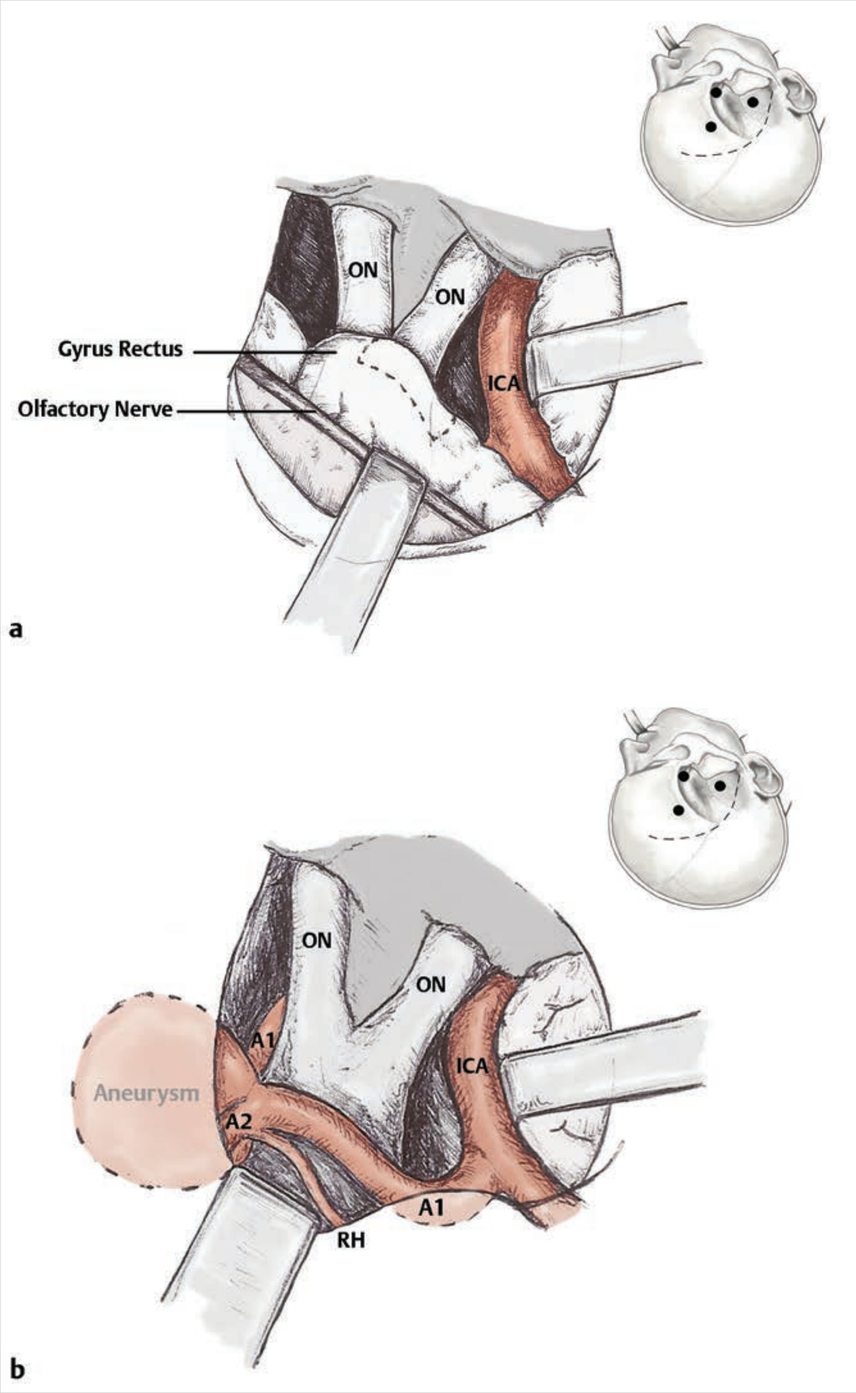

Fig. 8.2 Initial intradural exposure for an anterior communicating artery aneurysm. **(a)** The frontal lobe has been elevated to expose the optic nerves (ON). The carotico-optic cistern and sylvian fissure have been opened to expose the ipsilateral right internal carotid artery (ICA). The site of potential removal of the gyrus rectus is shown. **(b)** Post partial gyrus rectus resection revealing the ipsilateral right A1 and A2, the recurrent artery of Heubner (RH) as well as contralateral A1 and aneurysm neck.

Opening the sylvian fissure detaches the frontal and temporal lobes from each other, which greatly aids in frontal lobe retraction and exposes the proximal ipsilateral Al, facilitating early temporary clip application. In certain instances, the carotid bifurcation may be more distally located in the sylvian fissure and extensive dissection would be required. In such cases, however, there is usually significant looping of A1 inferiorly from the bifurcation and the more distal aspect of the ipsilateral A1 can be found where it crosses the optic nerve without opening the sylvian fissure. Likewise, in cases of subarachnoid hemorrhage (SAH) where the sylvian fissure is difficult to identify and dissect, attempted opening should be avoided.

With slight retraction of the frontal lobe and further arachnoid dissection, the ipsilateral A1 can be followed distally to where it crosses the optic nerve. At this stage of the dissection, the recurrent artery of Heubner is vulnerable to injury as it courses anterolateral to the A1 segment in 60% of cases.[3] Thus, it may be encountered before the A1 segment during the initial dissection and retraction of the frontal lobe. Moreover, the recurrent artery of Heubner should not be confused with the orbitofrontal artery, which is often the second major branch of the A2 segment and courses perpendicularly over the gyrus rectus and olfactory tract. The orbitofrontal artery may be important to identify because following it proximally can lead to the

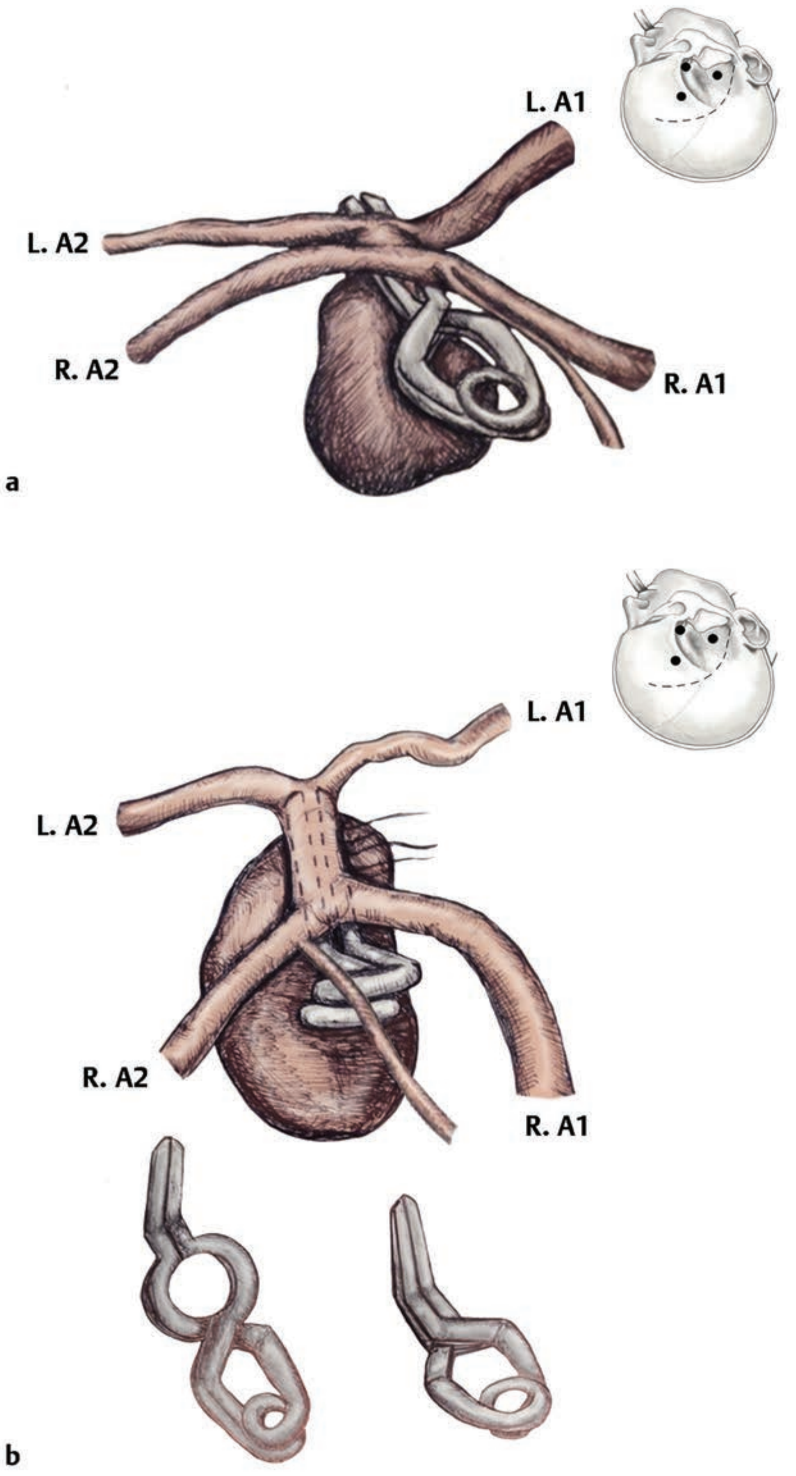

Fig. 8.3 Superiorly projecting aneurysms. **(a)** Schematic demonstrating a straight aneurysm clip used for anterior communicating complexes parallel to the skull base. **(b)** Schematic demonstrating angled or angled fenestrated aneurysm clips used for anterior communicating complexes perpendicular to the skull base. (*Continued*)

location of an obscured aneurysm neck. The gyrus rectus may be resected at this point. In our practice, resection is performed frequently, but only the minimum required to accomplish adequate exposure of the adjacent arteries and aneurysm. Gyrus rectus resection is accomplished by coagulating the pia of the gyrus rectus medial to the olfactory tract while protecting the orbitofrontal artery. The pia is then opened by sharp dissection and a small portion is carefully suctioned away while initially preserving the most medial pial layer to protect against damage of adjacent blood arteries and aneurysm. With a small retractor positioned in the resection bed, excellent visualization of the A1/A2 junction can be obtained (▶ Fig. 8.2b). Gyrus rectus resection may be unnecessary for inferiorly projecting aneurysms, ACoA–aneurysm complexes located close to the planum sphenoidale within the suprachiasmatic cistern, and in particularly relaxed brains.

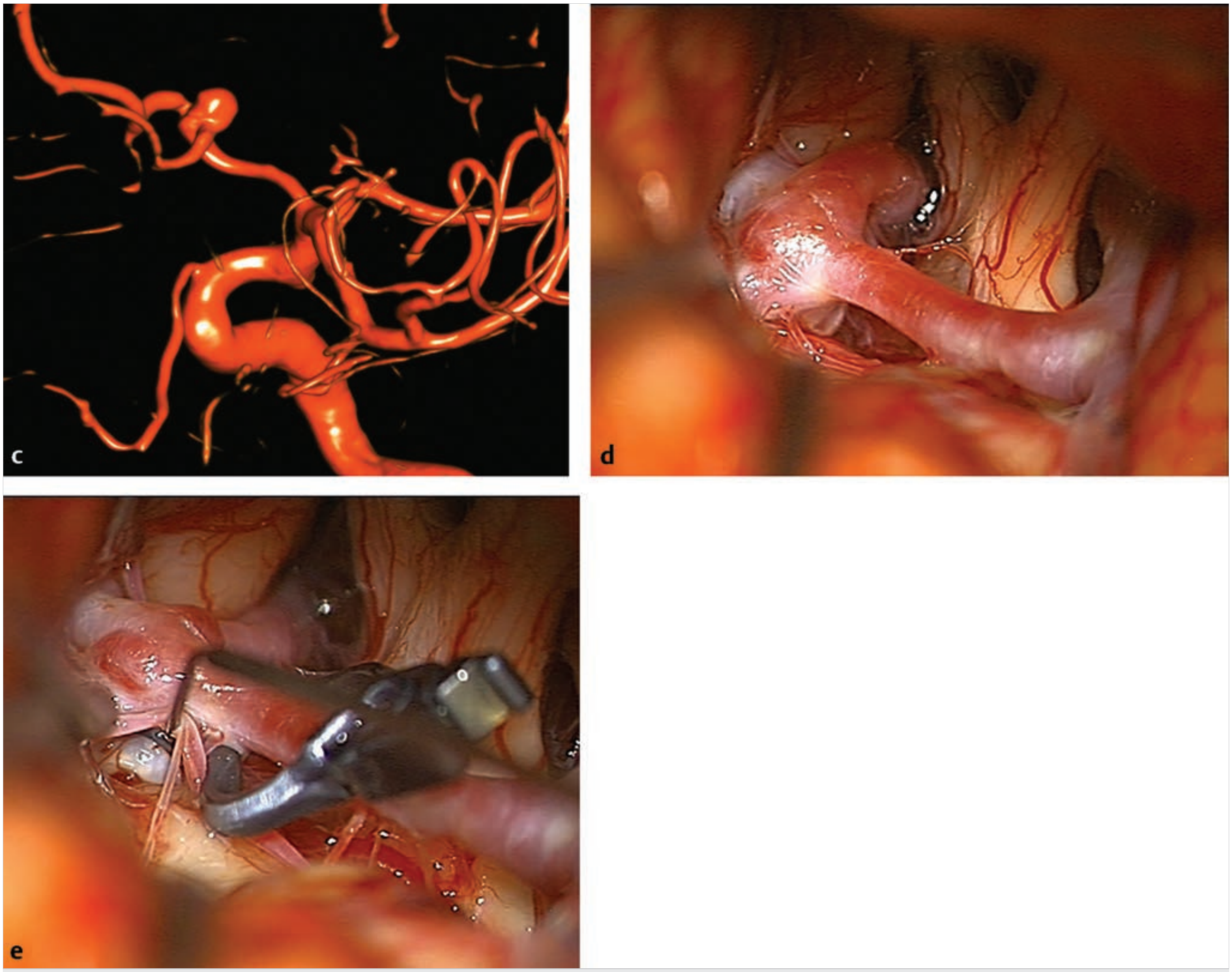

Fig. 8.3 (*Continued*) **(c)** Three-dimensional angiography demonstrating a superiorly projecting aneurysm. **(d)** Intraoperative photograph showing this aneurysm prior to aneurysm clip placement. **(e)** Intraoperative photograph showing the aneurysm after fenestrated aneurysm clip placement. Note the importance of evaluating the aneurysm complex circumferentially in order to fully visualize the aneurysm and perforating arteries.

Opening the interhemispheric fissure allows safer retraction of the ipsilateral frontal lobe. However, the anatomy of the aneurysm and the projection of the dome must be clearly understood prior to this dissection. Anteriorly and, especially, inferiorly directed aneurysms may be immediately beneath the arachnoid of the interhemispheric fissure and are prone to injury during this dissection. Conversely, extensive opening of the interhemispheric fissure is especially advantageous for superiorly projecting aneurysms located high within it.

In order to obtain sufficient control of the aneurysm during temporary clipping, control over the contralateral A1 is advisable, even if this artery appears hypoplastic or atretic on vascular imaging. In nearly all cases, there will be a contralateral A1 artery that will cause bleeding of a prematurely opened aneurysm if only the ipsilateral A1 is controlled. Such bleeding may still occur even with temporary clipping of both A1 s due to excellent collateral flow through the A2 arteries. Contralateral A1 ease of exposure depends on the configuration of the ACoA–aneurysm complex, specifically, the location where the contralateral A1 crosses the optic nerve. The location can vary, but is usually more medial than the surgeon might anticipate. Therefore, dissection should start medially and progress laterally in order to avoid overlooking a very medially located artery. Care must be taken to avoid confusing a large recurrent artery of Heubner for the contralateral A1. The relative orientation of the ACoA segment to the planum sphenoidale is another critical factor to evaluate. If the ACoA complex is parallel to the planum, both the ipsilateral and contralateral A1 and A2 may be visualized more easily. Conversely, if it is more perpendicular, the contralateral arteries will be obscured. During temporary clipping, the contralateral A1 clip should be placed first. A long, slightly curved clip may aid in clipping during this more technically difficult clip placement. Subsequently, the ipsilateral A1 can be clipped with a shorter clip in order to avoid obscuring the area of dissection.

The next step involves identification of both A2 arteries, if possible. Finding the ipsilateral A2 is essential and is usually accomplished by following the ipsilateral A1 distally and laterally, thereby exposing the region between the ipsilateral A2 and the aneurysm neck. At this point, a third, tapered retractor blade can

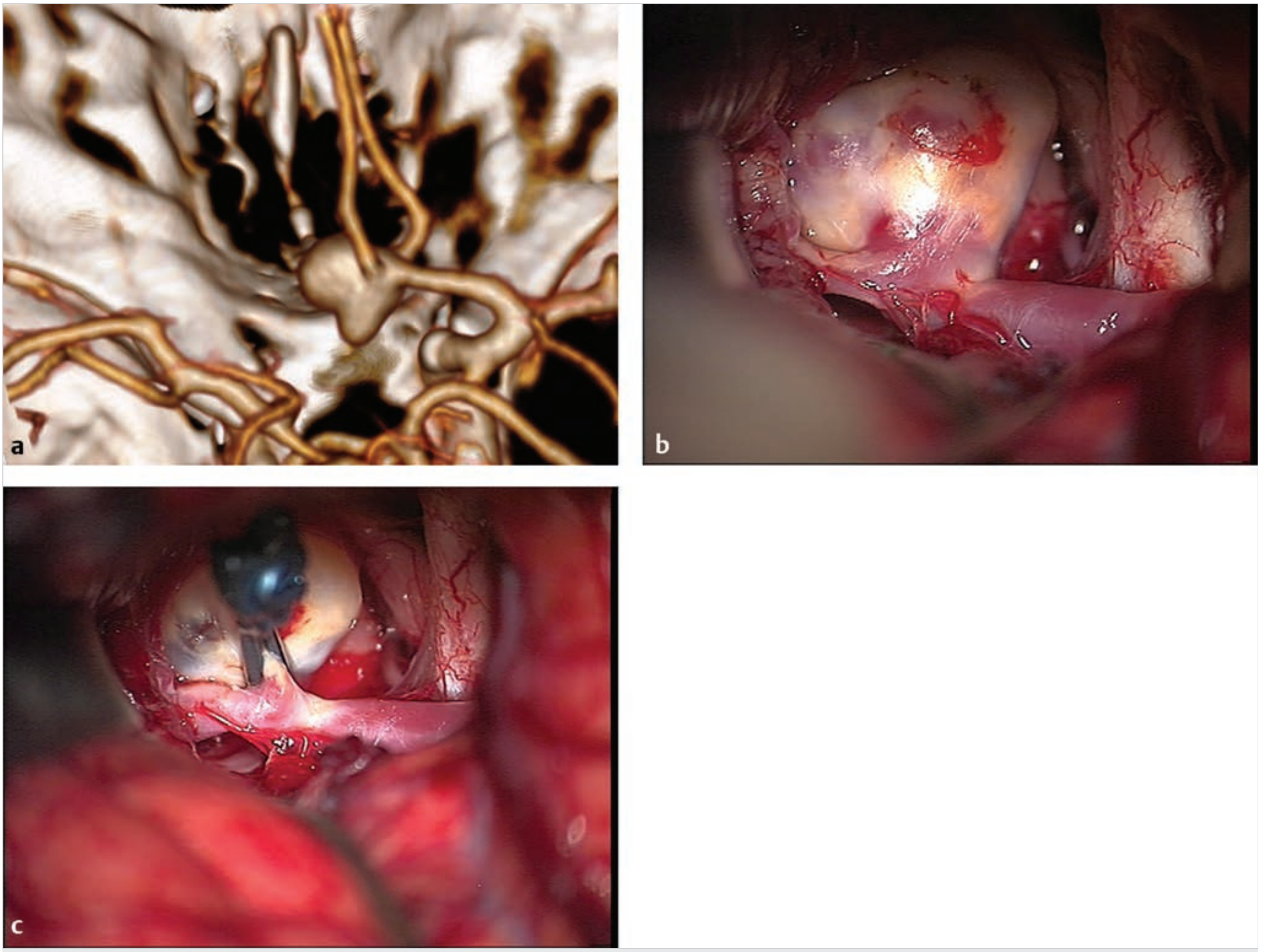

Fig. 8.4 Aneurysms with a combination of projections. **(a)** Three-dimensional CT angiography demonstrating a giant anterior communicating artery aneurysm projecting both anteriorly and inferiorly. **(b)** Intraoperative photograph showing this aneurysm prior to aneurysm clip placement. **(c)** Intraoperative photograph showing this aneurysm after aneurysm clip placement.

be used to retract the frontal lobe. The second retractor is located more proximally and maintains exposure of the ipsilateral Al. Though finding the contralateral A2 is always preferable, this may be very easy to nearly impossible depending on the anatomy. In some instances, the contralateral A2 may not be identifiable until the aneurysm has been clipped. Finally, the numerous perforating arteries arising in this region are extremely important. They should be visualized and protected (▶ Fig. 8.1, **Video 8.1**).

8.3.3 Clip Application

ACoA aneurysm necks are seldom ready to be clipped upon initial exposure. The aneurysm neck should be defined accurately. Once defined, cleavage planes need to be created between the aneurysm neck, adjacent arteries, and perforating arteries. Premature clip placement without full understanding of the global anatomy is strongly discouraged as it will likely lead to premature aneurysm rupture, inadequate aneurysm occlusion, and violation of the perforating arteries. Wide-necked aneurysms can be reconfigured with bipolar coagulation at a low setting with ample irrigation, but the origin of perforating arteries must be carefully protected from damage. Temporary clipping may be helpful. Though no defined guidelines regarding duration and quality of temporary clipping exist, in ACoA aneurysms we believe it should be avoided or kept to the minimum required to obtain a safe and adequate dissection and clipping of the aneurysm neck due to the large number of critical perforators in this region. If the dissection is performed over a dominant A1 and the aneurysm anatomy is relatively straightforward, temporary clipping of the ipsilateral A1 can be helpful to relax the aneurysm complex for safer dissection. In addition, if the anatomy is obscure and there is a high likelihood of opening the aneurysm prematurely, temporary clipping is advised. When a lengthy and difficult dissection is expected, placing a contralateral A1 temporary clip is a safe initial maneuver because an ipsilateral A1 temporary clip can be placed quickly if urgently needed. When prolonged temporary clipping is anticipated or deterioration of evoked potentials, if monitored, occurs, thiopental loading to achieve burst suppression on the electroencephalogram should be considered. Furthermore, the patient should be maintained with a normal to slightly hypertensive blood pressure during temporary clipping.

Again, aneurysm anatomy should be carefully evaluated prior to clipping. ACoA aneurysms occur in a variety of projections and configurations. Yasargil classified aneurysms according to their surgical orientation, and when adapted to an anatomical classification in relationship to the planum, as used in this text, his findings were that 34% projected superiorly, 23% anteriorly, 13% inferiorly, and 14% posteriorly, while 16% were complex, multilobulated aneurysms pointing in multiple directions (▶ Fig. 8.4a–c).[4] Clipping strategies vary depending on the projection of the aneurysm.

Superior

Superiorly projecting aneurysms are found between the A2 segments, embedded entirely within the interhemispheric fissure. Though temporary clip placement is relatively straightforward as the A1 arteries and ACoA are readily visible, placement of the permanent clip can be difficult and hazardous due to the close proximity of the infundibular and hypothalamic perforators to the posterior wall of the aneurysm. The orientation of the ACoA in relation to the planum sphenoidale is a significant factor in permanent clip selection. For a relatively perpendicular orientation, a simple straight clip with blades placed superior to the A1/A2 complex, both ipsilateral and contralateral, will suffice (▶ Fig. 8.3a). However, in most cases, the orientation is relatively parallel and a straight clip is not advocated due to the considerable amount of retraction required for placement. In such cases, either a 45-degree angled clip or a slightly angled fenestrated clip would necessitate much less retraction (▶ Fig. 8.3b). The angled clip should be placed with the blades superior to the A1/A2 complex. For the fenestrated clip, the fenestration typically encircles the ipsilateral A2, with the blades lying parallel to the ACoA and passing superiorly to the A1/A2 complex on the contralateral side (▶ Fig. 8.3c–e).

Anterior

Anteriorly projecting aneurysms can be favorable because the critical infundibular and hypothalamic perforators course in a direction opposite to the aneurysm. When these aneurysms are relatively small with a well-defined neck, obliteration can be fairly straightforward by using a curved clip placed between the two A2 arteries perpendicular to the ACoA (▶ Fig. 8.5a). However, in a large aneurysm with a wide neck, this maneuver can lead to kinking of the two A2 arteries. Additionally, in such cases, generation of a plane between the aneurysm and the A2 arteries, specifically on the contralateral side, may be difficult and lead to rupture of the aneurysm during dissection. Furthermore, the ACoA segment is enlarged in the anteroposterior plane. Inadequate clipping of the superior portion of the aneurysm neck

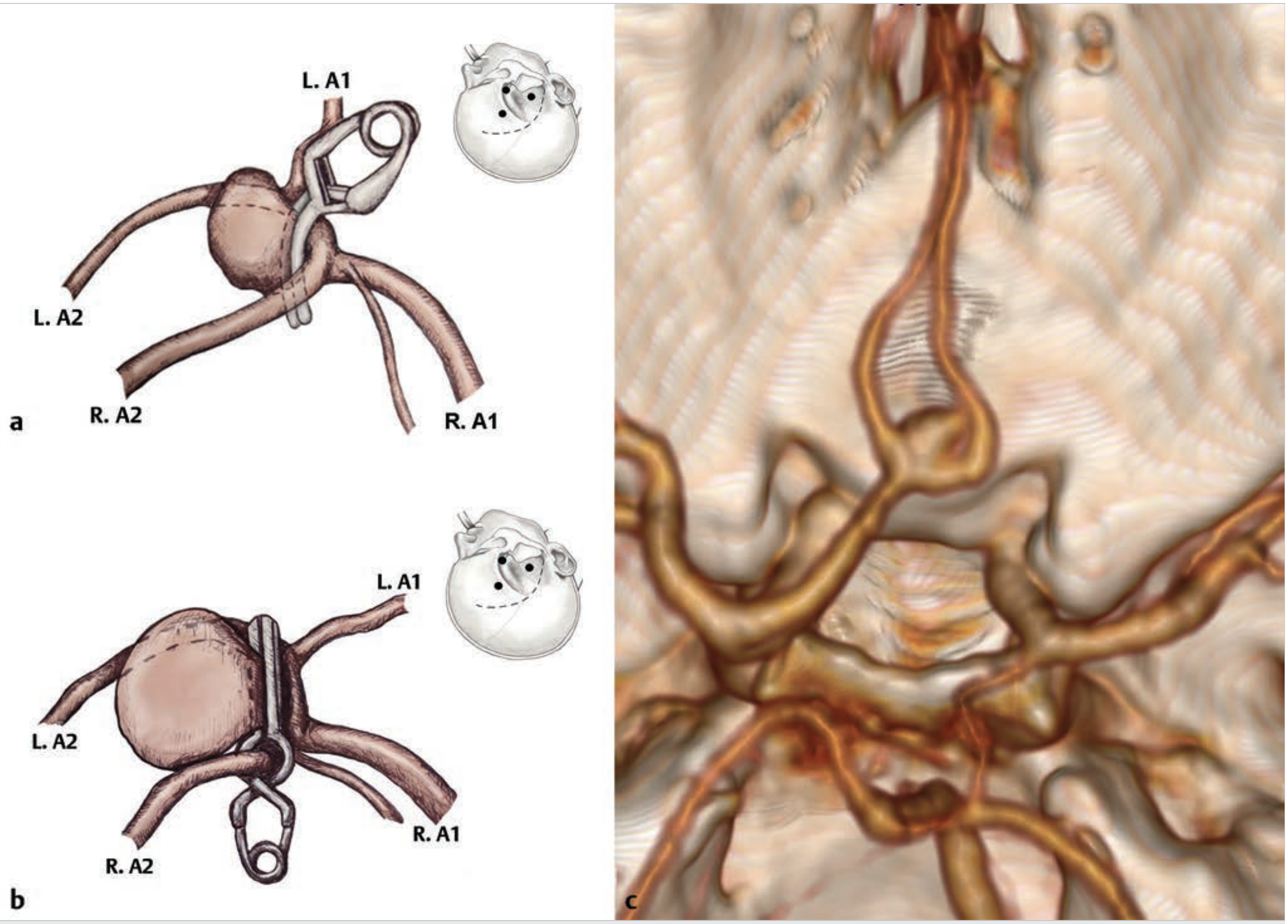

Fig. 8.5 Anteriorly projecting aneurysms. **(a)** Schematic demonstrating curved aneurysm clip placement. **(b)** Schematic demonstrating fenestrated clip placement. **(c)** Three-dimensional CT angiography demonstrating an anteriorly projecting aneurysm.

may occur due to poor visualization as well as underestimation of the neck size and extent of aneurysm dome projection superiorly. Thus, placement of a straight fenestrated clip parallel to the ACoA, encircling the ipsilateral A2 with the distal blades either lying superiorly or inferiorly to the A1/A2 contralateral complex, is advocated (▶ Fig. 8.5b). Dissection of both the superior and inferior aspects of the ipsilateral A1/A2 complex in order to visualize the aneurysm and its neck three-dimensionally is an extremely important principle. This maneuver is the only way to verify total aneurysm neck obliteration (▶ Fig. 8.5c–e, **Video 8.2**).

Inferior

The initial dissection and retraction of the frontal lobe must be performed with great care for inferiorly projecting aneurysms as they are vulnerable to premature rupture. The aneurysm dome may be adherent to the optic chiasm or skull base and can obscure the contralateral A1, leading to difficulty with temporary clipping. Furthermore, permanent clips often must be placed without prior identification of the contralateral A2 origin. Evaluation of contralateral A2 patency occasionally requires aneurysm decompression following final clip placement. The posterior wall of the aneurysm can typically be easily displaced anteriorly to visualize and separate the infundibular and hypothalamic perforators prior to application of the final clip. A straight aneurysm clip parallel to the ACoA, with blades passing inferior to the A1/A2 arteries, often will suffice (▶ Fig. 8.6a–d, **Video 8.3**).

Posterior

In our experience, small, isolated, posteriorly projecting aneurysms are rare. Posterior projection is usually found as part of a large, complex, multilobulated aneurysm. Similarly to inferiorly pointing aneurysms, they may be adherent to the optic chiasm and may obscure the contralateral A1 and A2. Additionally, they can be the difficult and treacherous to clip because the critical infundibular and hypothalamic perforators surround the neck and may be found either over the inferior wall or, less commonly, over the superior wall. Extensive dissection of the perforators and careful consideration of clip configuration are usually required (▶ Fig. 8.7).

Circumferential (ACoA Ectasia)

Although infrequently described in the literature, circumferential ectatic dilation of the ACoA segment with small blister-like aneurysms poses a significant surgical challenge and is more common than the literature indicates. The specific hemorrhage site may be impossible to secure with an aneurysm clip, while endovascular options are limited and challenging. Furthermore, these blister-like aneurysms frequently lead to pseudoaneurysm formation and, if not identified prior to clipping, may lead to catastrophic intraoperative hemorrhage. The only true treatment of such a diseased ACoA segment is complete isolation from the circulation by trapping with two aneurysm clips. Such an action can be performed safely if there are no perforating arteries originating from the posterior aspect of the ACoA segment and each A2 receives blood flow through its respective A1 (▶ Fig. 8.8a–d, **Video 8.4**).

Partially Coiled Aneurysms

Surgical clipping of partially coiled aneurysms is becoming increasingly common and the principles are similar to those for other aneurysms in this region and other partially coiled aneurysms.

8.3.4 Confirmation

Following final aneurysm clip placement, verification of complete aneurysm sac obliteration as well as establishment that flow in the bilateral A2 and perforating arteries has not been compromised is of utmost importance. As for other aneurysms, this is accomplished through meticulous microscopic inspection, intraoperative micro-Doppler flow evaluation, and

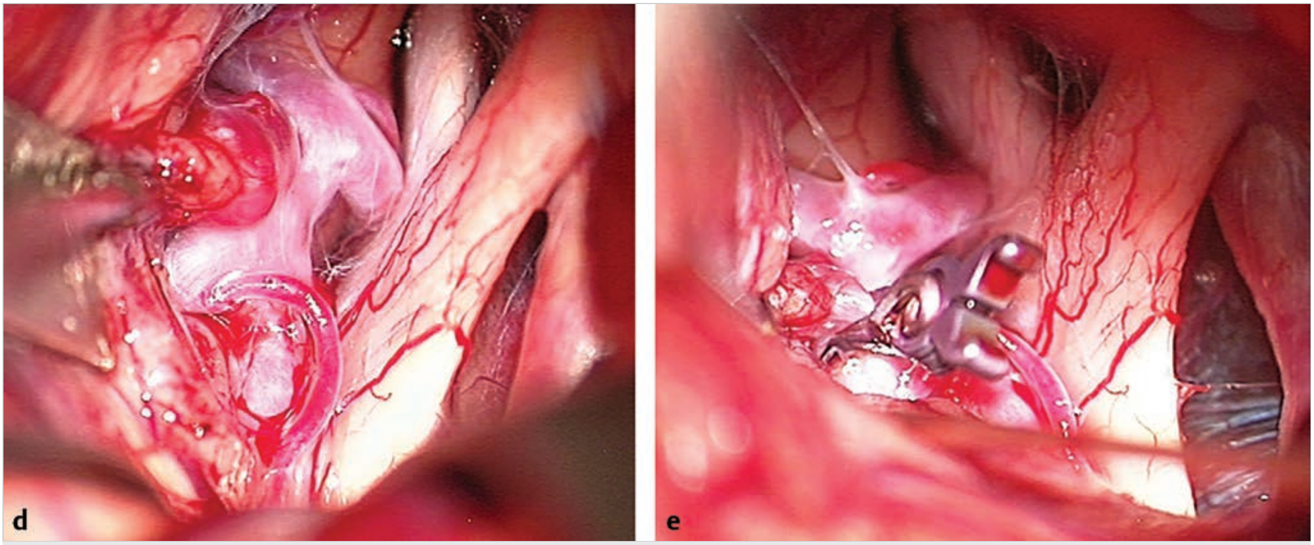

Fig. 8.5 (*Continued*) **(d)** Intraoperative photograph showing this aneurysm prior to aneurysm clip placement. **(e)** Intraoperative photograph showing this aneurysm after aneurysm clip placement.

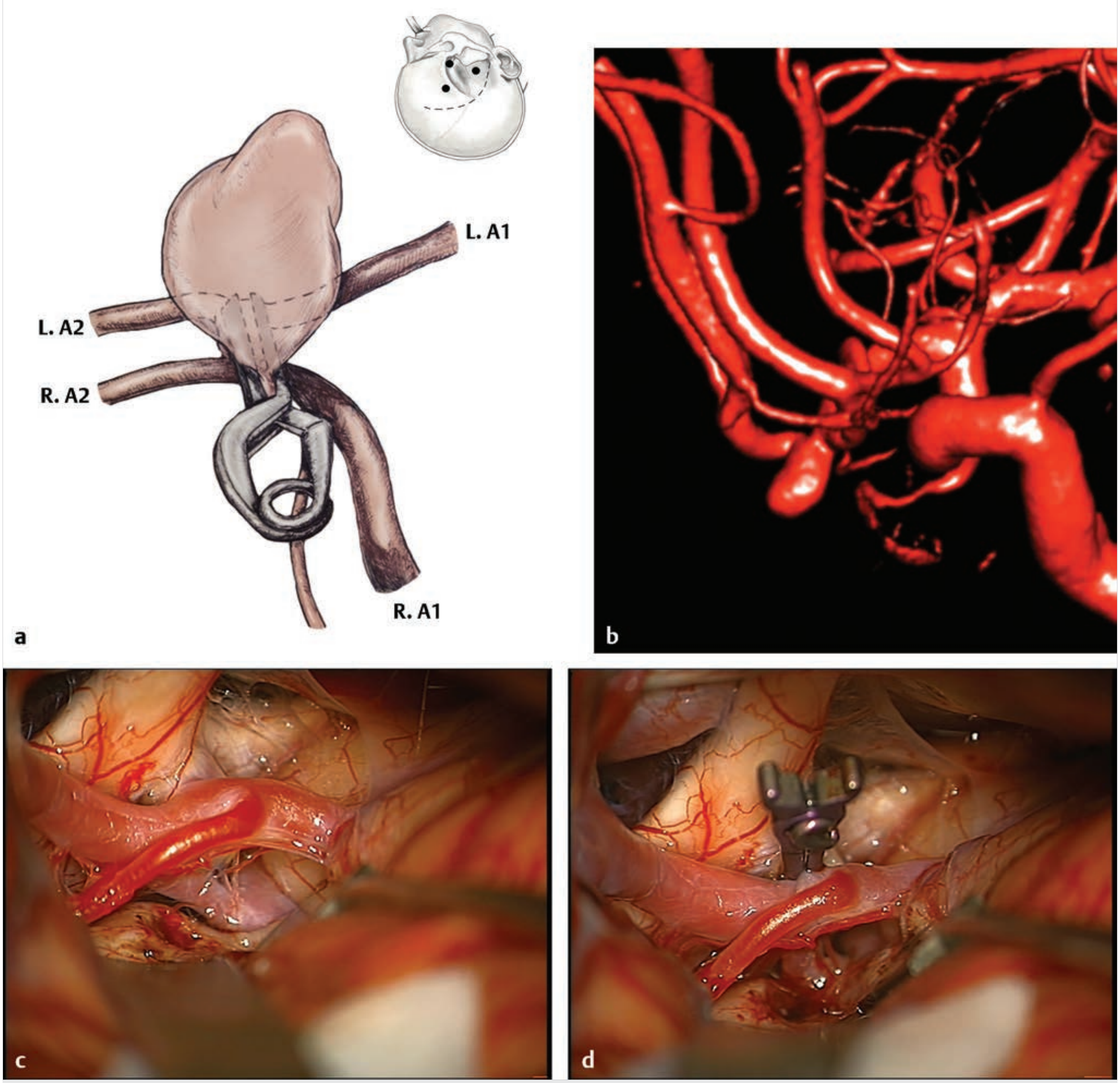

Fig. 8.6 Inferiorly projecting aneurysms. **(a)** Schematic demonstrating straight aneurysm clip placement. **(b)** Three-dimensional angiography demonstrating an inferiorly projecting aneurysm. **(c)** Intraoperative photograph showing this aneurysm prior to aneurysm clip placement. **(d)** Intraoperative photograph showing this aneurysm after aneurysm clip placement.

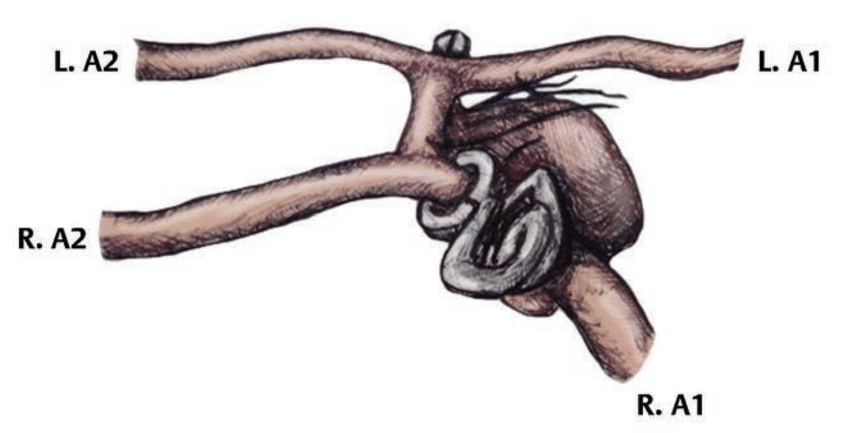

Fig. 8.7 Posteriorly projecting aneurysms. Schematic demonstrating potential clip placement for these rare aneurysms.

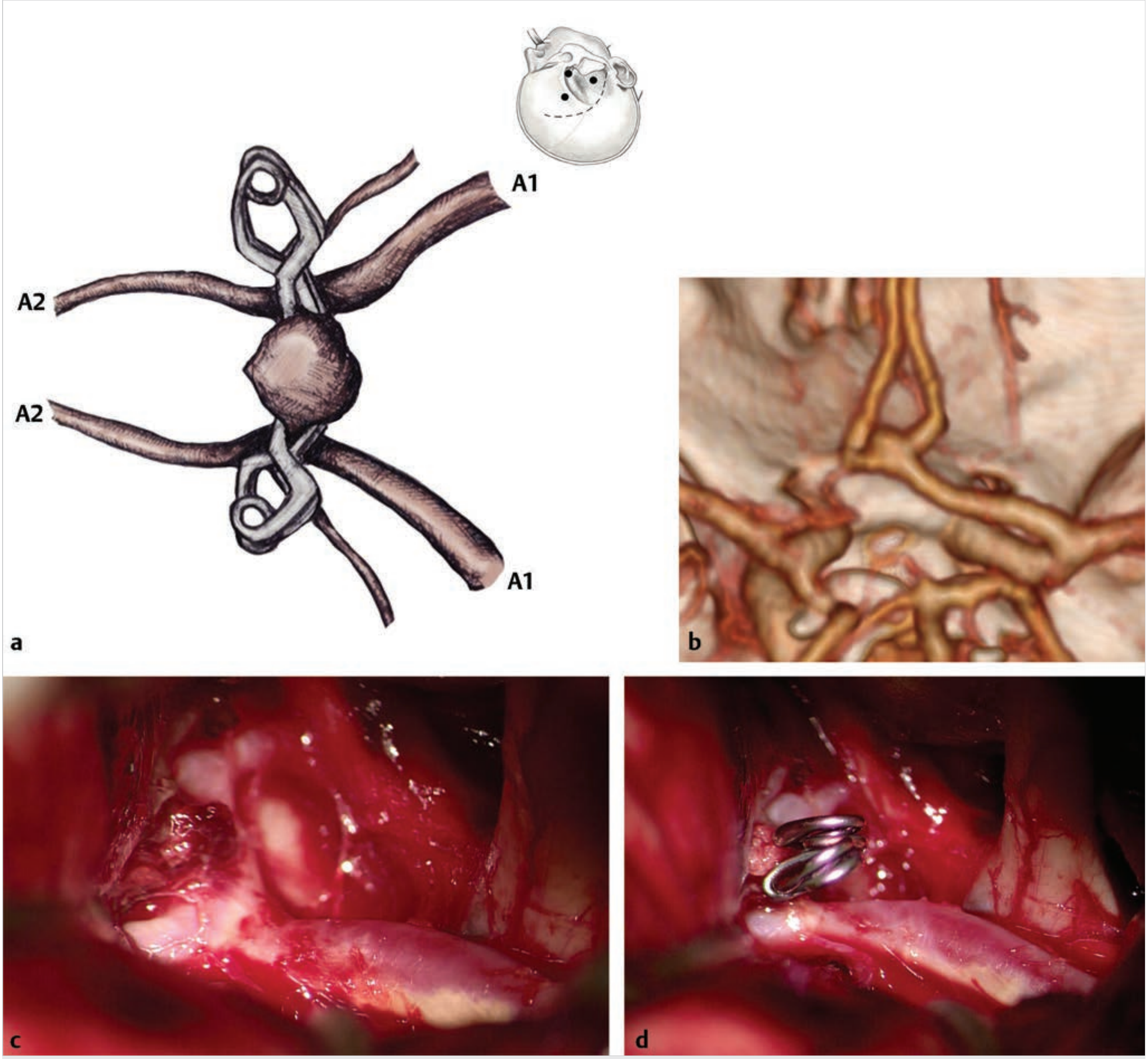

Fig. 8.8 Circumferential disease (ectasia) of an anterior communicating artery segment. **(a)** Schematic demonstrating trapping of the diseased, ectatic segment of the anterior communicating artery. **(b)** Three-dimensional CT angiography demonstrating an ectatic anterior communicating artery segment. **(c)** Intraoperative photograph showing the diseased anterior communicating artery segment prior to aneurysm clip placement. **(d)** Intraoperative photograph showing the diseased anterior communicating artery segment after aneurysm clip placement.

indocyanine green angiography in all cases. Intraoperative catheter angiography is not routinely used in our institution, but is reserved for complex and giant aneurysms. In addition, it should be understood that occlusion of even major perforating arteries does not necessarily lead to motor or somatosensory evoked potential changes and evaluation of their patency relies heavily on the previously stated modalities.

In cases of severe SAH, the lamina terminalis is frequently opened and a cisternal drain is positioned in the chiasmatic cistern inferior to the ACoA. Closure is performed in the standard fashion. Postoperative computed tomography angiography is obtained in all cases, as well as, if indicated, catheter angiography.

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9 Middle Cerebral Artery Aneurysms

R. Loch Macdonald

Abstract
The middle cerebral artery (MCA) is the largest of the cerebral arteries, and aneurysms along its course constitute about 20% of all ruptured intracranial aneurysms. Despite its highly complex anatomy, the MCA lends itself favorably to surgery due to the relative proximity of most aneurysms to the lateral cerebral convexity and the often but not universally accepted difficulty treating these aneurysms endovascularly. That being said, surgery for MCA aneurysms is particularly unforgiving in that occlusion of the MCA branches is associated with infarction, with clinical deficits in more than 50% of cases.

Keywords: aneurysm, craniotomy, middle cerebral artery

9.1 Patient Selection

The middle cerebral artery (MCA) originates as the largest terminal branch of the internal carotid artery, the other major branch being the anterior cerebral artery.[1,2] The MCA origin is lateral to the optic chiasm, posterior to the medial and lateral olfactory striae, and inferior to the anterior perforated substance, and lies in the sphenoidal compartment of the sylvian fissure. The first (M1) or sphenoidal segment of the MCA courses laterally posterior and parallel to the sphenoid ridge. Ml bifurcates into superior and inferior trunks that are the M2 segments. Branches of Ml include lenticulostriate arteries, which perforate the anterior perforated substance, and there are usually cortical branches, most commonly the anterior temporal and polar temporal arteries.

The anatomy of the MCA and the aneurysm should be reviewed. The direction of projection, size, and morphology (saccular, fusiform, dissecting, infectious, traumatic, proximity to and potential adherence of MCA branches, presence of atherosclerosis, calcifications, and thrombosis) of the aneurysm should be scrutinized on three-dimensional catheter or computed tomography (CT) angiography. For the most common aneurysm of the MCA, which is located at the MCA bifurcation, it is important to determine the length of the Ml segment preoperatively, which, on the anteroposterior angiogram view, shows the depth of the aneurysm. The lenticulostriate branches nearly always originate along the superior and posterior parts of the MCA; thus, the arachnoid should be opened and the Ml manipulated on the frontal and inferior sides.

Most MCA aneurysms are diagnosed following subarachnoid hemorrhage (SAH) and/or intracerebral hemorrhage.[1,2] They also are the most common incidentally found aneurysm. Rarely, giant MCA aneurysms present with symptoms related to mass effect, ischemia, or seizures (▶ Fig. 9.1, ▶ Fig. 9.2, ▶ Fig. 9.3).

9.2 Indications and Contraindications for Surgery

The main indications for surgery are for ruptured or symptomatic MCA aneurysms. In patients with SAH, we aim to secure the aneurysm as early as possible after SAH to minimize the risk of further hemorrhage and also before the onset of angiographic vasospasm and delayed cerebral ischemia. Emergent treatment may be indicated in cases where there is a sizable intracerebral hematoma that needs to be evacuated to improve the neurological condition (▶ Fig. 9.4). In general, the authors treat all patients with ruptured aneurysms except those who have bilaterally fixed pupils or worse, and those in whom recovery is deemed highly unlikely. The decision to adopt conservative treatment assumes that poor neurological status is not due to factors such as intracerebral hematoma or hydrocephalus. If evacuation of an intracerebral hematoma is necessary, the aneurysm has to be secured at the same surgery.

The urgency of the situation will dictate whether a catheter angiogram can be performed prior to surgery. In a patient with an intracerebral hematoma that needs to be removed acutely, a CT angiography can give adequate information on the location and size of the aneurysm so that surgery can be performed. We rely heavily on CT angiography and reserve catheter angiography for complex or giant aneurysms and those where endovascular treatment is contemplated, such as unruptured aneurysms or those with favorable morphology and unfavorable surgical characteristics (advanced patient age, poor medical risk, neck calcification, severe atherosclerosis, etc.). The next indication is to secure unruptured aneurysms in the case where the risk of aneurysm rupture over time is considered to exceed surgical risk.

9.3 Alternative Considerations to Surgery

The alternatives to surgery are endovascular treatment or no treatment.[3] No treatment for the aneurysm may be appropriate in poor-grade patients after SAH or for unruptured aneurysms. Management of unruptured aneurysms is controversial, and the surgeon must be convinced that the risk of morbidity and mortality from surgery is lower than that due to the natural history of the aneurysm if left alone.[4,5]

Once a decision has been made to secure an aneurysm, the next consideration is whether clipping or coiling is more appropriate. MCA aneurysms tend to have a wide neck with branches arising at the base, which has generally made them more suitable for clipping. Endovascular technology is advancing, although efficacy studies of new devices have rarely been done. Management decisions have to be made on a case-by-case basis.

9.4 Risks

Specific risks associated with craniotomy for MCA aneurysm clipping include intraoperative aneurysm rupture, incomplete aneurysm occlusion, seizures, occlusion of significant branches or perforators with clip application leading to varying degrees of neurological deficit, and injury to the frontalis branch of the

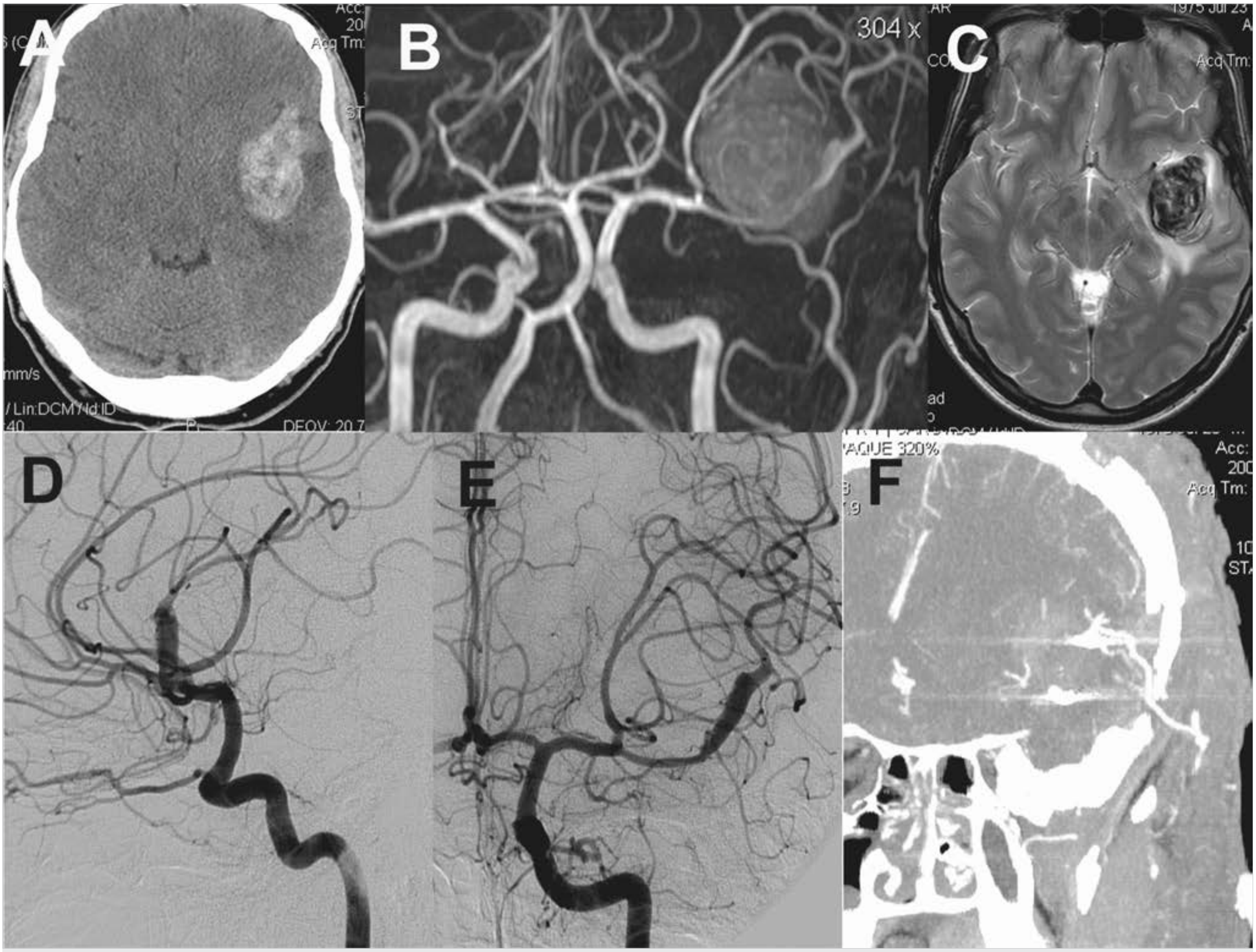

Fig. 9.1 A giant thrombosed middle cerebral artery aneurysm that arises from the left inferior M2 branch can be seen on **(a)** cranial computed tomography, **(b)** magnetic resonance angiography, and **(c)** T2-weighted magnetic resonance imaging scans. The patient was a 32-year-old man who presented with a seizure. Lateral **(d)** and anterior posterior **(e)** catheter angiography shows the aneurysm arising from the inferior M2 trunk. **(f)** The aneurysm was treated by trapping and bypass by suturing the superficial temporal artery to the M2 branch distal to the aneurysm.

facial nerve. Occlusion of MCA branches can lead to motor weakness, expressive or receptive dysphasia, higher-functioning disturbance, visual field defects, parietal signs, and even death due to brain swelling or severe neurological deficit.

9.5 Preoperative Preparation

9.5.1 Medications

Nimodipine is commenced after diagnosis in patients with SAH.[6] The usual oral dose is 60 mg every 4 hours. Maintenance of normovolemia is imperative in the perioperative phase. There is no uniform agreement on choice of fluid replacement, but our practice is to use 0.9% NaCl with close monitoring of electrolytes and sodium and potassium supplementation as necessary. Anticonvulsants such as phenytoin or levetiracetam may be administered to patients who have had a seizure, who are at high risk of having one (intracerebral hematoma), or who would be potentially harmed by one (poor-grade patients, those with already increased intracranial pressure).

9.5.2 Other Factors

Smooth induction of anesthesia without altering the blood pressure is important so as to reduce the risk of aneurysm rupture or rerupture. The head pins should only be applied when it can be assured that the patient is adequately anesthetized. We do not use lumbar drainage but place an external ventricular drain (EVD) preoperatively in patients with neurological symptoms and signs due to hydrocephalus or intraoperatively via Paine's point when additional brain relaxation is needed.[7] An arterial line and urinary catheter are used in all patients. Central venous lines are optional. Always be prepared for massive uncontrolled bleeding. Intraoperative hypothermia was not beneficial in a large, randomized study, so despite experimental data to the contrary, its use has to be considered questionable at this time.

For complex or giant aneurysms where prolonged temporary clipping or bypass procedures may be necessary, we use electroencephalography monitoring so we can induce burst suppression if necessary (▶ Fig. 9.1, ▶ Fig. 9.2, ▶ Fig. 9.3).

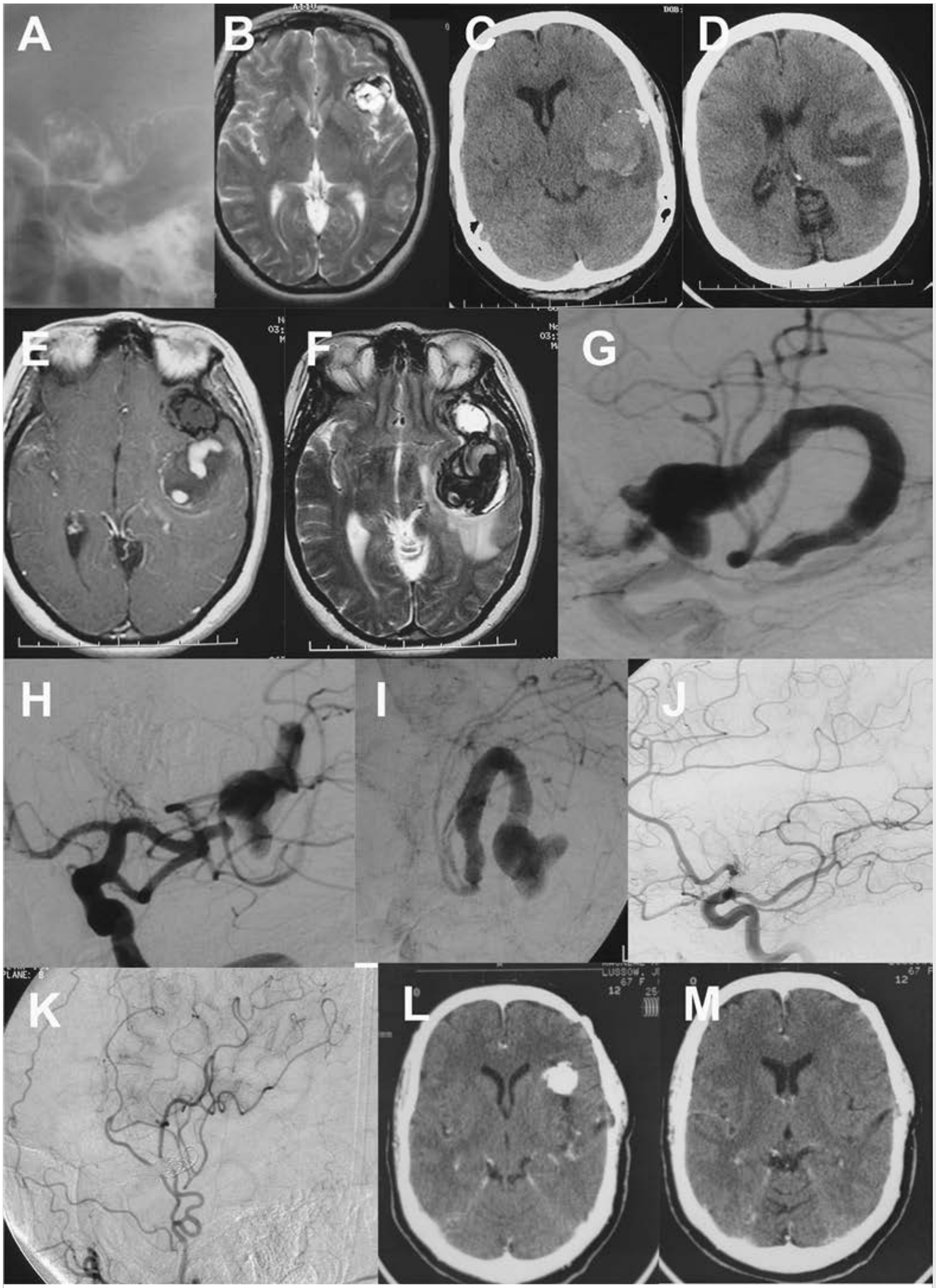

Fig. 9.2 A giant fusiform middle cerebral artery (MCA) aneurysm. **(a)** The aneurysm was found incidentally on a skull radiograph, and the initial lateral carotid angiogram shows the calcified part of the aneurysm with filling of MCA branches beyond the aneurysm. **(b)** A magnetic resonance study at that time shows the aneurysm in the left sylvian fissure. The 49-year-old woman was followed; 10 years later, she presented with headaches and dysphasia, and a computed tomography (CT) scan **(c, d)** showed marked enlargement of the aneurysm with intra-aneurysmal thrombosis and edema in the brain surrounding it. **(e)** Axial T1-weighted gadolinium-enhanced and **(f)** axial T2 images show the giant, thrombosed aneurysm with the suggestion of a serpentine channel through the aneurysm. **(g)** A left internal carotid catheter angiography lateral view shows the channel through the aneurysm with three distal MCA branches emerging from the distal end of the aneurysm. Sequential oblique views early **(h)** and late **(i)** arterial phase show that the three branches are on the deep side of the aneurysm sac and course up to the surface over the top of the aneurysm. The aneurysm was treated by bypassing two superficial temporal artery branches into two of the MCA branches and then filling the proximal end of the aneurysm inflow with coils. **(j)** Postoperative angiogram 6 months later shows occlusion of the aneurysm with coils on the lateral view of the internal carotid injection and **(k)** revascularization distally of the MCA. The CT scan **(l, m)** shows resolution of much of the aneurysm mass. The patient recovered fully and remained well at last follow-up 4 years later.

9.6 Operative Procedure

9.6.1 Positioning

The patient is positioned supine with a rolled sheet under the ipsilateral shoulder to allow neck rotation (**Video 9.1**). The bed is flexed and put in a 20-degree reverse Trendelenburg position. A three- or four-pin radiolucent head holder is applied. The normal angle from the anterior clinoid process to the pterion is just over 45 degrees. For MCA aneurysms, turning the patient's head 45 degrees to the contralateral side means the operative pathway will be almost vertically downward along the sphenoid ridge. Intraoperative neuronavigation can be used to guide positioning and, if required, EVD insertion and locating where an intracerebral hemorrhage presents closest to the brain surface. This reduces the need for retraction of

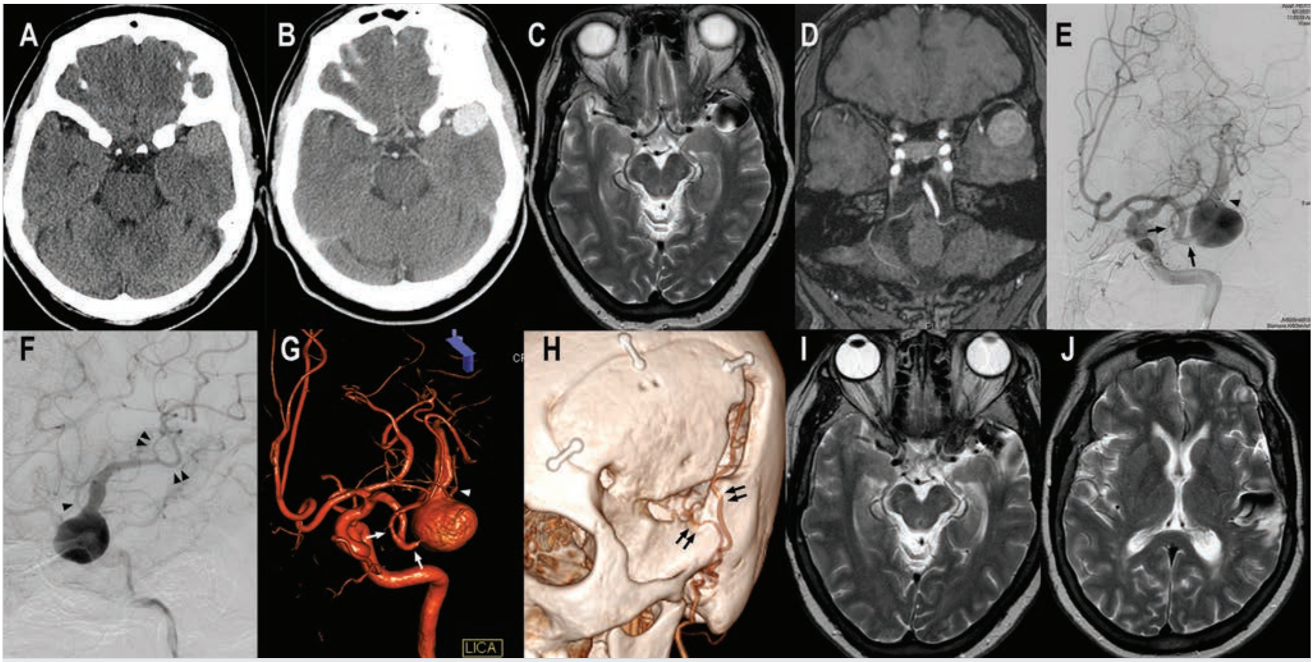

Fig. 9.3 A 68-year-old woman with type 2 diabetes and hypertension presented with multiple syncopal episodes and was found to have a giant fusiform left middle cerebral artery aneurysm on plain **(a)** and enhanced **(b)** axial CT scans and axial T2-weighted **(c)** and time-of-flight magnetic resonance imaging angiography **(d)**. Catheter angiography **(e, f)** with a 3D reconstruction **(g)** demonstrates the arterial inflow from the M1 segment of the middle cerebral artery and a branch arising from M1 proximal to the aneurysm. A dilated portion of the middle cerebral artery emanates from the saccular part of the aneurysm giving rise to two branches (*double arrowheads*) as well as another branch directly from the sac (*arrowhead*). The patient underwent trapping of the aneurysm and bypass by anastomosis of one superficial temporal artery branch into the middle cerebral artery branch arising directly from the sac (*arrowhead*) and a second into the larger distal branch (*double arrowheads*). The distal clip tapping the aneurysm was placed proximal to the bifurcation of the two distal branches to allow filling of both through the one anastomosis. Postoperative CT angiography shows the two bypasses (*double arrows*, **h**). Postoperative T2-weighted magnetic resonance imaging shows collapse of the aneurysm and preservation of the brain parenchyma **(i, j)**. The patient recovered fully.

the temporal lobe, and often only the frontal lobe needs to be retracted. The head is also extended to allow the frontal lobe to fall away naturally once dissection has begun. Finally, the rotated, extended head is elevated upward to facilitate venous return. In this position, the malar eminence is the highest point. The head is then fixed. Mannitol, 1 g/kg, and furosemide (20–40 mg) are given intravenously in most cases with SAH except in cases such as an elderly person with minimal SAH where brain relaxation will not be needed.

9.6.2 Skin Incision and Craniotomy

The authors use a standard pterional craniotomy. We only shave a line of hair along the incision and a line along where an EVD might exit posteriorly. Once the patient has been positioned and draped (standard craniotomy drape), an incision is begun 1 cm anterior to the tragus at the level of the zygoma extending initially vertically and slightly posteriorly and then gently curving over the superior temporal line to end at the hairline just across the midline. This preserves the superficial temporal artery so that it can be used for intraoperative angiography. The temporalis muscle is reflected in a single layer with the scalp flap, which makes it easier to preserve the frontalis branch of the facial nerve. The temporalis fascia can be cut with a knife so it does not shrink down if cut by cautery. The muscle can be divided with cautery. Dissection of the muscle off the bone should preserve the periosteal layer attached to the muscle. A cuff of fascia and muscle along the superior temporal line can be left behind to reapproximate the muscle to the region of the superior temporal line with sutures at the end of the procedure.

The musculocutaneous flap is covered with saline-soaked gauze and retracted with fish hooks that can be attached to the carbon fiber basal frame. A free bone flap is fashioned with up to three burr holes joined up with a craniotome. The first burr hole is at the keyhole above the frontozygomatic suture and inferior to the superior temporal line. Additional burr holes can be made just above the posterior end of the zygomatic arch at the lower end of the skin incision and above the superior temporal line at the posterior margin of the scalp incision. If after removing the bone flap the dura is tense, additional brain relaxation can be achieved by mild hyperventilation. Also, we insert an EVD into the frontal horn of the lateral ventricle once the dura is open.[7] This is done by aiming perpendicularly to the brain from the top of a triangle 2.5 cm back along the sylvian fissure and 2.5 cm superiorly.

Further bone removal is then directed at removing the lateral one-third of the sphenoid wing using either a high-speed drill or rongeurs. For an anteriorly directed MCA aneurysm, be aware that the aneurysm could be adherent to the dura of the sphenoid ridge. A clue to this is if there is subdural blood on the CT scan, suggesting the aneurysm has eroded through the arachnoid. Early retraction of the brain away from the dura can precipitate early intraoperative rupture in these cases.

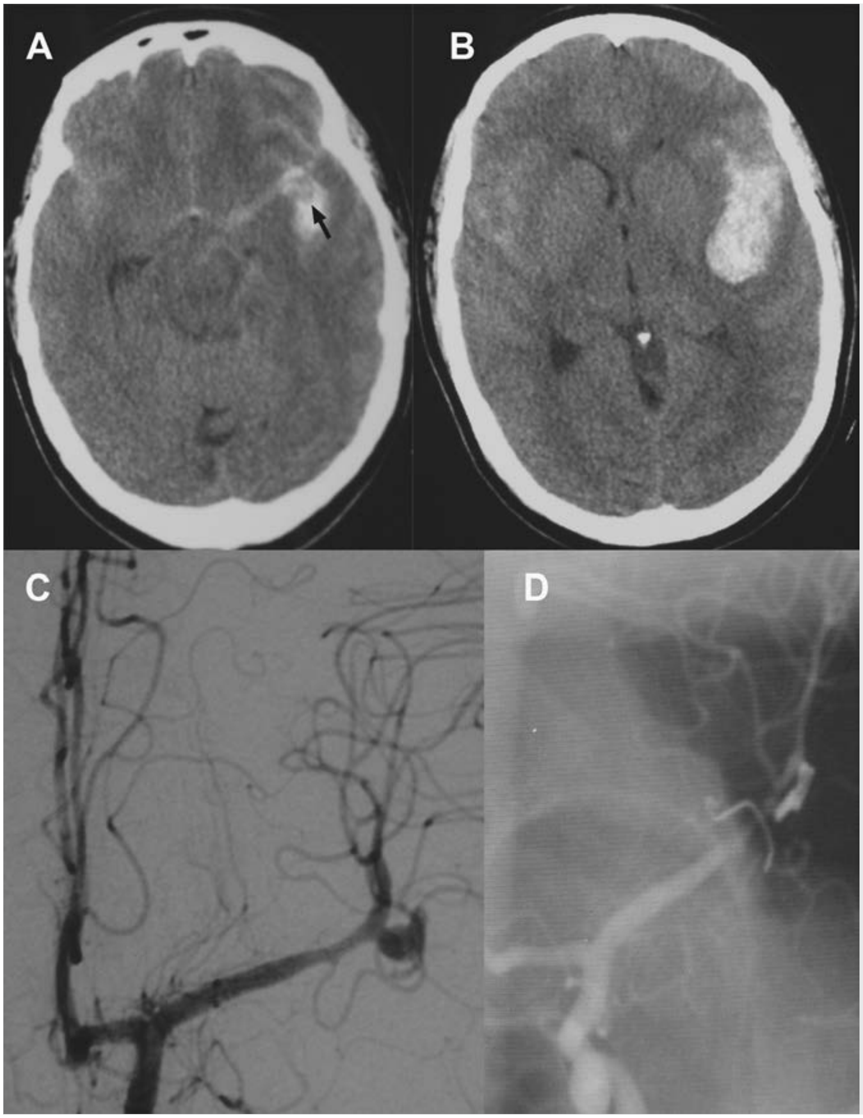

Fig. 9.4 **(a)** Cranial computed tomography showing subarachnoid hemorrhage and **(b)** a hematoma in the temporal lobe on the left. The aneurysm can be seen in the sylvian fissure (*arrow*). **(c)** Angiography shows the aneurysm, which **(d)** was clipped uneventfully.

The dura is opened in a **U**- or **C**-shaped fashion and retracted toward the musculocutaneous flap. Extradural bleeding can be controlled with standard tack-up stitches.

9.6.3 Operative Approach

The operation proceeds under the operating microscope (**Video 9.1**). There are three main approaches to MCA aneurysms. The transcortical approach is through the superior temporal gyrus. The two transsylvian approaches are opening of the sylvian fissure from medial to lateral or from lateral to medial. The author almost exclusively uses a lateral to medial transsylvian approach. The main disadvantage of this approach is that the aneurysm may be encountered before proximal control is obtained.

The superior temporal gyrus approach is usually for cases where there is a large intracerebral hematoma. In these cases, we make a cortical incision in the anterior part of this gyrus, enter the clot cavity, and suction out some of the clot. The aneurysm is anterior so we suction out enough clot away from the aneurysm to achieve brain relaxation and then return to open the sylvian fissure. The rest of the clot is removed after the aneurysm is clipped, either through the cortical incision or via the pathway where the aneurysm ruptured through into the brain. The medial to lateral transsylvian approach involves elevation of the frontal lobe, identification of the optic nerve, entry into the chiasmatic and carotid cisterns, and dissection of the sylvian fissure from proximal to distal. The advantage is early proximal control, but it requires greater retraction of both frontal and temporal lobes. This is probably preferable to the lateral to medial approach other than for unruptured aneurysms or maybe until one gains confidence in microdissection.

For the lateral to medial approach, the arachnoid over the fissure is opened sharply with an arachnoid knife starting 2 to 3 cm posterior to the sphenoid ridge (▶ Fig. 9.5 and ▶ Fig. 9.6). The sylvian veins tend to run along the surface of the temporal lobe, so dissection should begin on the frontal side of these veins. As one proceeds deeper, retraction of the edges of the fissure with a patty and suction tip allows sharp dissection of bridging fibers. Self-retaining tapered retractors can be used. Cortical veins that bridge the fissure can be coagulated and divided. Once an M3 or M2 branch is identified, it can be followed

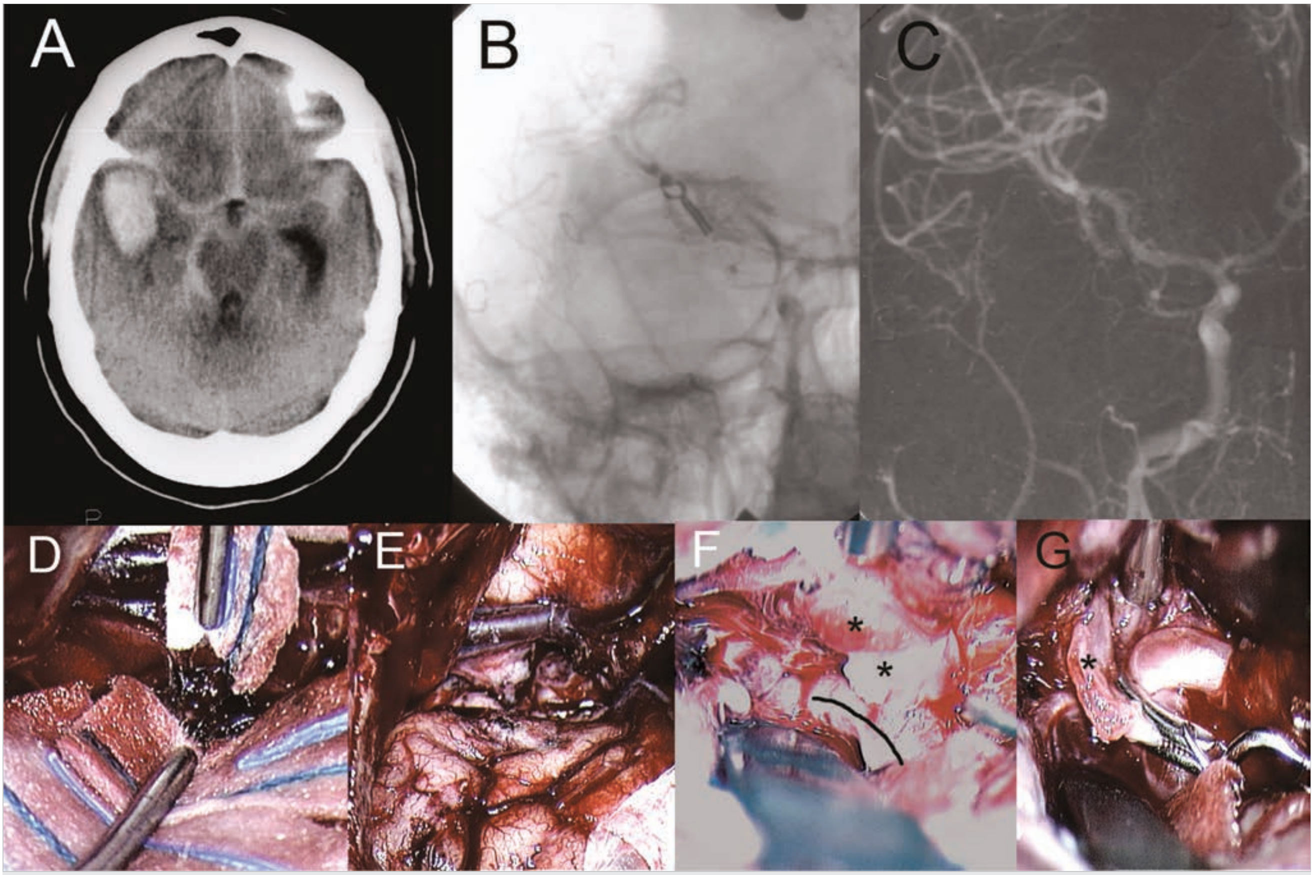

Fig. 9.5 **(a)** A cranial computed tomography scan of a ruptured middle cerebral artery aneurysm showing subarachnoid hemorrhage (SAH) and a hematoma in the temporal lobe. The patient was taken to surgery immediately and the aneurysm exposed and clipped, as shown by intraoperative angiography unsubtracted **(b)** and subtracted **(c)** views performed by retrograde injection of the superficial temporal artery. **(d)** Initial exposure of the sylvian fissure using a lateral to medial approach shows the SAH upon opening of the arachnoid over the lateral part of the fissure. **(e)** The clot is suctioned away and a distal M2 branch is identified. **(f)** This is followed proximally to the aneurysm (*asterisk*), with the course of the middle cerebral artery shown by the curved dark lines. **(g)** The aneurysm is clipped with a clip that curves along the neck of the aneurysm. The collapsed sac of the aneurysm is seen distal to the clip (*asterisk*).

proximally to the MCA bifurcation. Subarachnoid hematoma can be suctioned away. The arachnoid can also be cut with microscissors. Once the MCA bifurcation is identified, the next step depends on the location of the aneurysm. If the aneurysm is at the MCA bifurcation, then it will have been seen and the next step is to get proximal control by exposing the Ml. This is done by leaving the aneurysm dome undisturbed as much as possible and dissecting near the base of the aneurysm to expose the proximal artery. If the aneurysm is along the M1 segment, further dissection is necessary and is best along the anterior and inferior sides of the Ml.

9.6.4 Preparation and Clipping

Once the aneurysm has been identified and proximal exposure is adequate, the aneurysm has to be prepared for clipping. The region of the aneurysm should be carefully inspected to identify any branches that may be draped over the aneurysm and also any branches or perforators that may be compromised with clip application. This is achieved by visualizing the aneurysm neck in all directions, which sometimes requires dissection off the surrounding brain, sometimes including complete mobilization of the aneurysm. An important principle is that there are always at least two branches to find in addition to the proximal M1. Temporary clipping may soften the aneurysm and reduce the risk of rupture during dissection. Branches running along the aneurysm can usually be dissected off with a blunt dissector but occasionally may require sharp dissection for adhesions. For larger aneurysms, it may be very difficult to dissect branches off the aneurysm, and it may be better to use creative clipping strategies.

Once the clip is applied, the aneurysm should be reexamined to ensure that the clip is not compromising any branches. We generally confirm this by intraoperative indocyanine green angiography or angiography by retrograde cannulation and injection through the superficial temporal artery.

If the aneurysm ruptures prior to clip application, several strategies can be employed. If the point of rupture is small, then the suction tip over a small patty for a few minutes may be sufficient. Alternatively, a temporary clip can be applied proximally to the Ml and distally to the main branches if bleeding remains uncontrolled. This will then allow the aneurysm to be clipped successfully while it is isolated from the circulation. Any period of temporary occlusion should be minimized and consideration should be given to burst suppression if more than 10 minutes of occlusion is anticipated. Other situations where

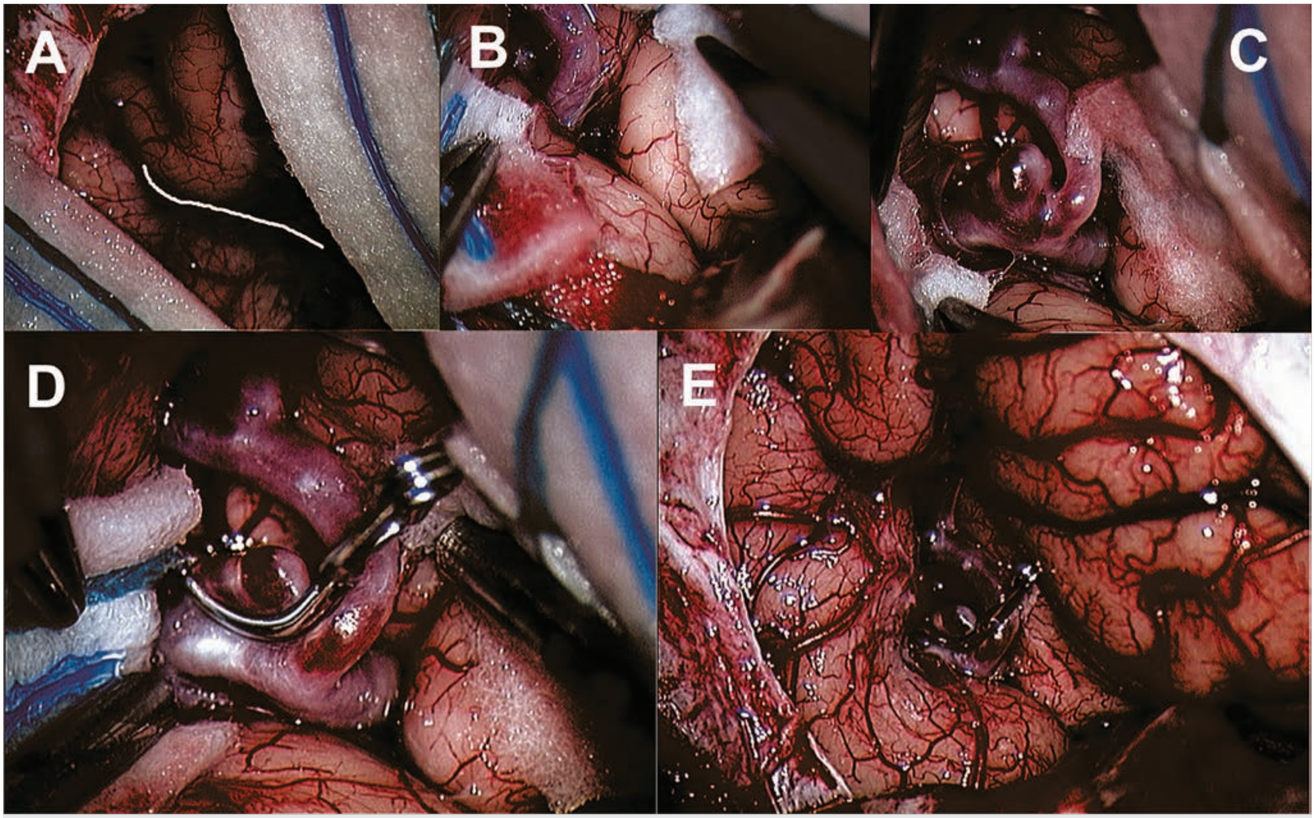

Fig. 9.6 Series of photographs of the dissection of the right sylvian fissure and exposure of a middle cerebral artery aneurysm. **(a)** The initial exposure with a line indicating the fissure. **(b)** The arachnoid has been opened and a distal M2 branch identified. **(c)** The initial view of the aneurysm shows that the neck is **V**-shaped and goes down into the fork between the M2 branches. **(d)** Because of this, clip reconstruction uses a curved clip that obliterates the aneurysm with no neck remnant and with preservation of the distal branches. **(e)** View at the end of the procedure.

temporary clip application may be necessary are atherosclerotic or calcified and large or giant aneurysms. Opening of the aneurysm to remove calcified or atherosclerotic material may be necessary to ensure proper clip placement and closure of the blades. This may be done with a dissector, but an ultrasonic aspirator may also be used, so have it ready in advance. Rupture at the time of clip application is dealt with by slowly closing the clip. If the rupture is distal to the clip and there is still bleeding, there may be incomplete occlusion of the aneurysm and a second clip may be necessary. If the bleeding is proximal to the clip, a second clip should be applied proximal to the first to gain control, after which the patency of the main vessel and branches can be assessed. In patients with giant aneurysms, removal of mass effect may be as important as clipping the aneurysm, depending on the patient's symptoms.

9.6.5 Closure

A nonabsorbable multifilament suture is used to close the dura in a continuous watertight fashion (**Video 9.1**). Central tacking sutures are placed from the dura through the bone, and the bone flap is replaced with titanium plates and screws. The temporalis fascia is then sutured along the line of earlier incision with 3–0 absorbable multifilament suture. If a cuff of fascia was left superiorly, this can also be reapproximated. The galea is closed with the same interrupted sutures and the skin with a running subcuticular 4–0 absorbable suture.

9.7 Postoperative Management Including Complications

The patient is awakened and extubated if they were sufficiently alert to do so preoperatively and if there were no intraoperative catastrophes that might render the patient unsafe for extubation. Neurological examination is performed to detect any untoward effects of surgery. SAH patients are monitored in intensive care for fluid balance to maintain normovolemia and permissive hypertension. Any change in neurological condition warrants laboratory investigations to ascertain the cause and usually an immediate CT scan. We do not routinely perform cerebral angiography after clipping MCA aneurysms but rely on intraoperative studies that are done in most cases.

Postoperative complications are listed under the section "Risks." Intraoperative angiography prevents inadvertent arterial occlusions and undipped residual aneurysms. Seizures are treated with anticonvulsants. Risk of postoperative stroke is also minimized by avoiding as much as possible manipulation of the arteries and temporary clipping.

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10 Distal Anterior Cerebral Artery Aneurysms: Anterior Interhemispheric Approach

Jason A. Ellis, Nikita G. Alexiades, Robert A. Solomon, and E. Sander Connolly Jr.

Abstract

Aneurysms arising from the anterior cerebral artery (ACA) distal to the anterior communicating artery account for between 1.5 and 9.0% of all intracranial aneurysms. The vast majority of these so-called distal anterior cerebral artery (DACA) aneurysms are found at or near the origin of the callosomarginal artery in the region of the genu of the corpus callosum. DACA aneurysms are frequently associated with anomalous ACA anatomy, multiple aneurysms in 40 to 55% of patients, and traumatic or mycotic etiologies, and also tend to rupture at smaller sizes than aneurysms in other locations. The microsurgical treatment of DACA aneurysms presents a number of unique challenges stemming from their anatomic location. In his classic treatise on microneurosurgery, Yasargil summarized these challenges as follows: (1) The interhemispheric fissure and the callosal cistern are very narrow; (2) The depth of the falx may be short and, therefore, both cingulate gyri may be densely adherent; (3) Aneurysms at this location are commonly broad-based and frequently sclerotic. They involve the origins of the branching arteries to a varying degree; (4) Sclerosis of both the fundus of the aneurysm and the opposite pericallosal artery may occur at a site of attachment between the two, and this can lead to considerable difficulty in dissection; (5) At times, it will be quite difficult to tell from which ACA the aneurysm is arising; (6) The dome of the aneurysm may be densely fixed on the pia layer of the cingulate gyrus, or it may even be within the gyrus; (7) The aneurysms may be located at the bifurcation of an azygos A2.

Keywords: pericallosal artery, callosomarginal artery, anterior cerebral artery, intracranial aneurysm, interhemispheric, subarachnoid hemorrhage

10.1 Clinical Presentation and Diagnostic Workup

In the same manner as with aneurysms in other locations, distal anterior cerebral artery (DACA) aneurysms may present symptomatically with subarachnoid hemorrhage (SAH) or may be found incidentally.[1-5] When presenting with SAH, blood on computed tomography (CT) is most often prominent within the interhemispheric fissure with layering above the corpus callosum (▶ Fig. 10.1). This hemorrhage pattern may also be observed with superiorly directed anterior communicating artery (ACOM) aneurysms, potentially creating some diagnostic ambiguity. Depending on the dome orientation, DACA aneurysms may rupture into the ipsilateral or contralateral frontal lobe and/or into the

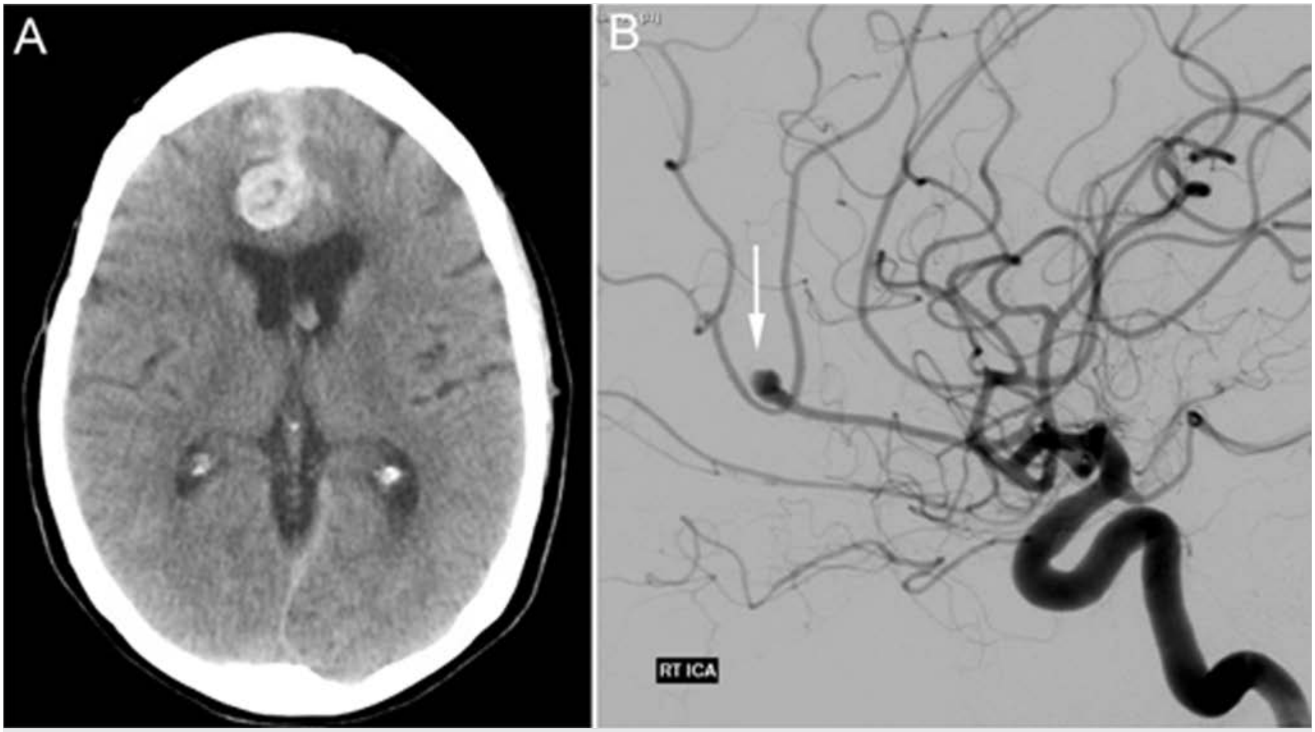

Fig. 10.1 This patient presented with a complaint of "worst headache of life" and progressed to obtundation. Head CT demonstrated subarachnoid hemorrhage within the anterior interhemispheric fissure as well as intracerebral hemorrhage within the right medial frontal lobe **(a)**. Catheter angiography revealed the bleed source to be a 5-mm pericallosal artery aneurysm (*arrow*) arising just distal to the branching of the callosomarginal artery (**b**).

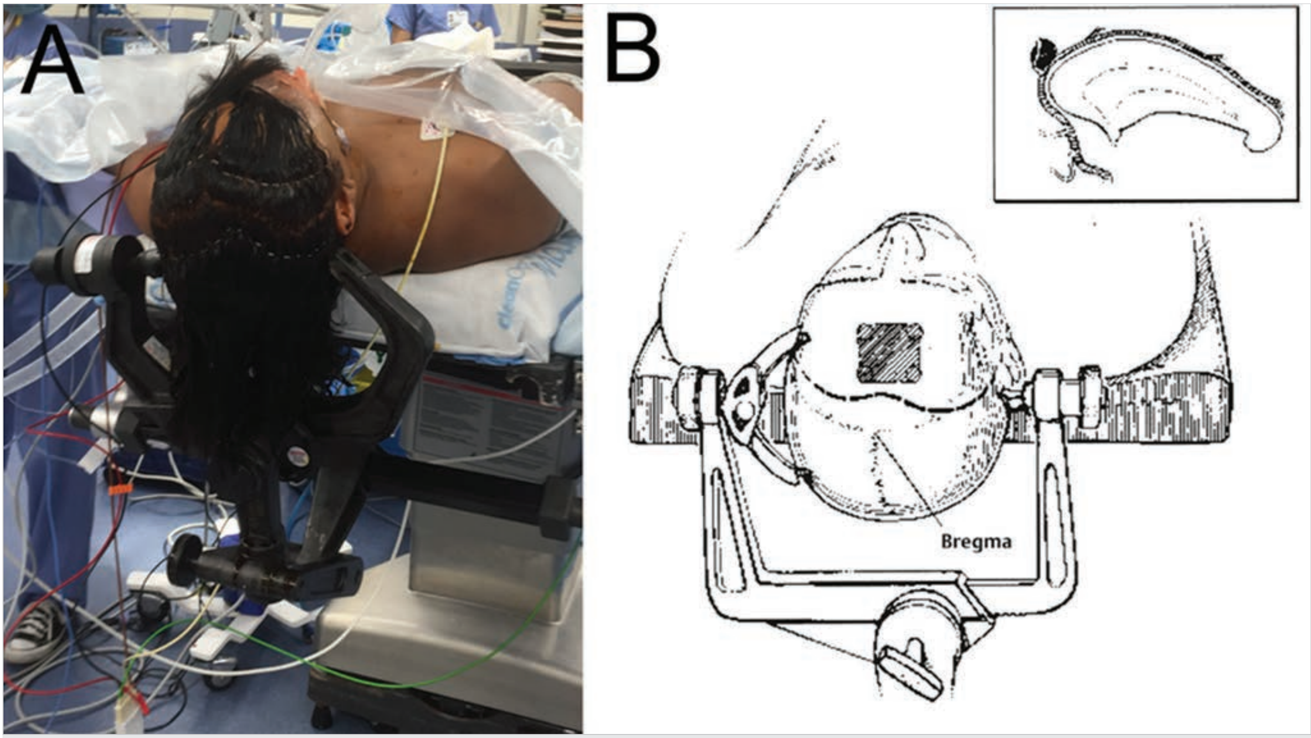

Fig. 10.2 For the anterior interhemispheric approach, the patient is positioned supine with the head pinned in a neutral position **(a)**. Note that the pins are placed sufficiently posterior so that they do not interfere with the planned bicoronal incision. The artist depiction illustrates an optimal neutral head position for approaching this A3 aneurysm (inset) in the region of the corpus callosum genu (**b**). Such aneurysms often arise just distal to the origin of the callosomarginal artery.

ventricles.[6,7,8] Less commonly, patients may present with seizures due to irritation of the underlying cortex or thromboembolism in the anterior cerebral artery (ACA) territory.

Cerebral vascular imaging with CT and/or catheter angiography is necessary for aneurysm characterization prior to operative intervention. Although catheter angiography remains the goal standard imaging modality, axial imaging may have additional utility for mapping bridging veins and craniotomy planning. Since the aneurysm dome is not uncommonly buried within one of the medial cerebral hemispheres, magnetic resonance imaging may enhance appreciation of the aneurysm–brain interface. Although not mandatory, consideration may be given to obtaining volumetric imaging so that frameless stereotaxy can be utilized.[9]

10.2 Treatment Options

Historically, DACA aneurysms have been most effectively treated with microsurgical clipping as opposed to endovascular coiling due to their peripheral location, small size, and unfavorable neck-to-dome and neck-to-parent artery ratios.[1] These aneurysms, when ruptured, may also result in a “blowout” at the bifurcation from which they arise, thus making endovascular repair more difficult or impossible without adjuncts such as balloon remodeling or stents.[4]

Given that patients with DACA aneurysms frequently harbor multiple aneurysms, consideration may be given to treating more than one aneurysm at the same time. It is our opinion that such an approach is most reasonable when multiple aneurysms can be accessed from the same surgical approach. We generally avoid treating additional unruptured aneurysms if a completely separate approach is needed so as to avoid increased operative times and additional brain manipulation. Both our experience and the literature suggest that the rupture risk of unsecured, unruptured aneurysms is not increased in the setting of hemodynamic or endovascular therapy for angiographic vasospasm and delayed cerebral ischemia.[10]

10.3 Anterior Interhemispheric Approach

10.3.1 Position

Although all aneurysms at and distal to the ACOM can be approached interhemispherically, those greater than 2 cm distal to the communicating artery must invariably be attacked in this manner. The patient is positioned supine with the head pinned neutral in a three-point skull clamp (▶ Fig. 10.2). When the aneurysm lies below or proximal to the genu of the corpus callosum, modest head extension may facilitate the approach. Intraoperative neuronavigation can be valuable for planning the trajectory to the aneurysm as well as finding it during surgery. Slight head flexion may be beneficial for approaching aneurysms arising from the A4 or A5 segments of the pericallosal artery. Although not favored by our group, the lateral position is also reasonable.

10.3.2 Skin Incision and Craniotomy

A bicoronal scalp incision, behind the hairline, extending from the ipsilateral zygoma across the midline to the contralateral superior temporal line is used. The temporalis fascia and muscle

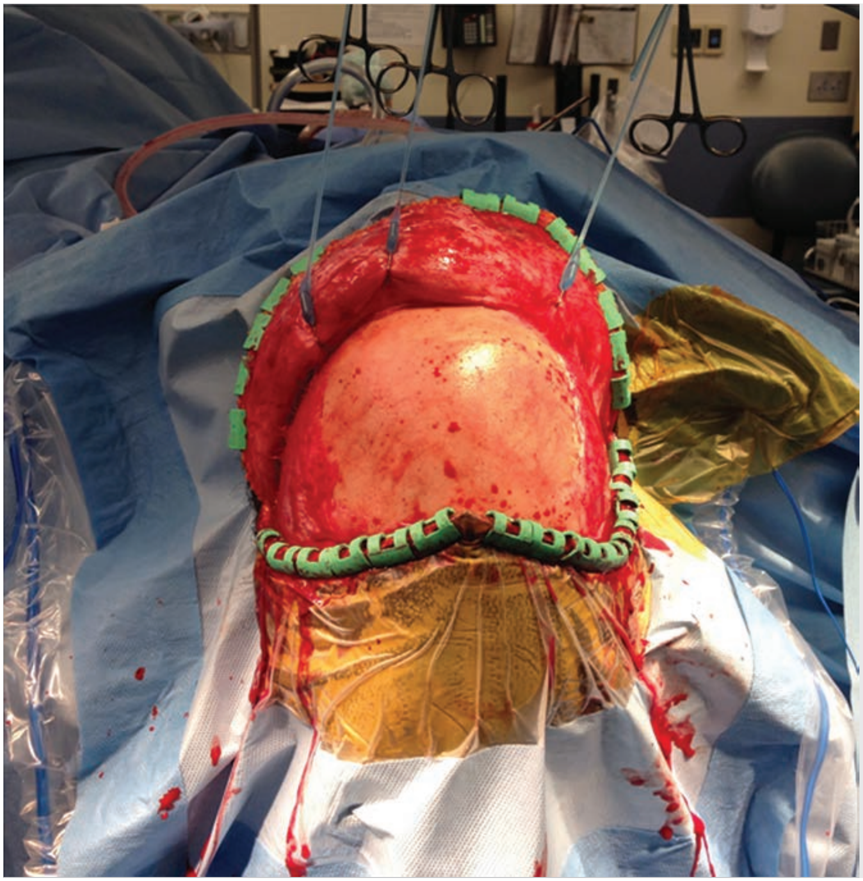

Fig. 10.3 This bicoronal scalp flap is retracted anteriorly over the orbital rim and held in place with hooks. Care is taken to ensure that no pressure is placed on the globe during scalp retraction. Note that the temporalis muscle is left down.

should be left down and intact (▶ Fig. 10.3). Care is taken to avoid pressure on the eyes while the scalp is flapped anteriorly. The exact rostrocaudal location of the proposed craniotomy is determined by the location of the aneurysm and of bridging veins to the superior sagittal sinus. We favor a nondominant, right-sided approach in most cases since DACA aneurysms from either ACA can generally be accessed below the falx from either side. Left-handedness, a left-sided frontal hematoma, or aberrant ACA anatomy may favor left-sided approaches.[8]

A parasagittal craniotomy is planned taking care to avoid violation of the frontal sinus when possible. The craniotomy extends from the ipsilateral superior temporal line and to just across the superior sagittal sinus on the contralateral side. To accomplish this, we burr slots across the full width of the sagittal sinus. A bone flap can thus be raised with the footplate attachment of the drill never contacting the sinus. In general, large flaps are created to enable flexibility in finding additional interhemispheric corridors should the bridging venous anatomy be unfavorable. Although it is classic teaching that a single bridging vein to the anterior superior sagittal sinus (anterior to the coronal suture) can be sacrificed with impunity, this maneuver should be avoided if possible.[11] Venous infarction is largely unpredictable when bridging veins are sacrificed, although they may be silent in the anterior frontal lobes.

10.3.3 Dural Opening

The dura is opened such that a flap based on the superior sagittal sinus can be reflected to the contralateral side (▶ Fig. 10.4a). Occasionally, cortical veins will enter the dura early, prior to bridging toward the sagittal sinus necessitating that a cuff of dura be left attached to the brain and not flapped contralaterally. Arachnoid adhesions to the dura can typically be dissected free, allowing the falx to be identified. Slight tension on the dural flap allows some contralateral retraction of the sagittal sinus which improves exposure of the interhemispheric fissure. Care must be taken not to occlude the sinus with this maneuver.

10.3.4 Microsurgical Dissection

After dural opening, a self-retaining retractor system is set up and the operating microscope is brought into the field. The cortical surface of the exposed frontal lobe is covered with cottonoids and gently dissected from the falx. Small, medially bridging veins below the level of the sinus are cauterized and cut. A 2- to 3-cm anteroposterior working corridor is developed and self-retaining retractors are placed on both the medial frontal cortex and, if necessary, the falx at its inferior margin. This facilitates exposure of the cingulate gyri bilaterally, which are dissected sharply using an arachnoid knife and microscissors (▶ Fig. 10.4b–c). The anterior-to-posterior working space between the cingulate gyri is progressively expanded. Subpial dissection should be avoided at this stage to avoid bleeding and brain tissue transgression.

At the base of the cingulate gyri lies arachnoid connections beneath which the cistern of the corpus callosum is located (▶ Fig. 10.5a). Within this cistern are the pericallosal arteries that in the setting of SAH are often encased in clot. Both arteries must be identified and invariably followed proximally. Unless a

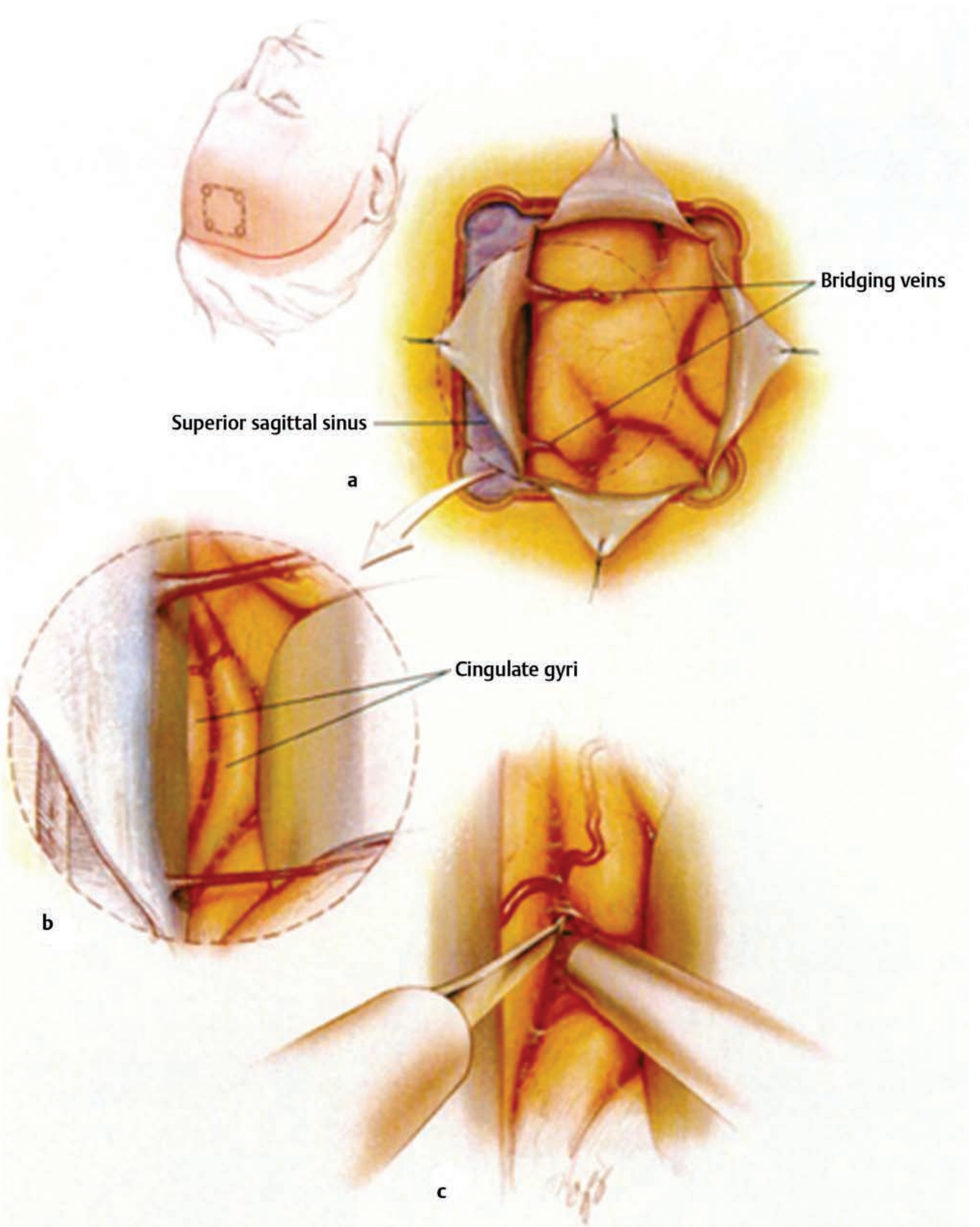

Fig. 10.4 The dura is flap opened contralaterally across the superior sagittal sinus **(a)**. Care is taken to preserve bridging veins when possible. Retractors are placed on both the medial frontal cortex and, if necessary, the falx at its inferior margin to facilitate exposure of the cingulate gyri **(b)**. A 2- to 3-cm working space is usually sufficient. Since the cingulate gyri are often tightly apposed, further sharp dissection is necessary to enter the callosal cistern **(c)**.

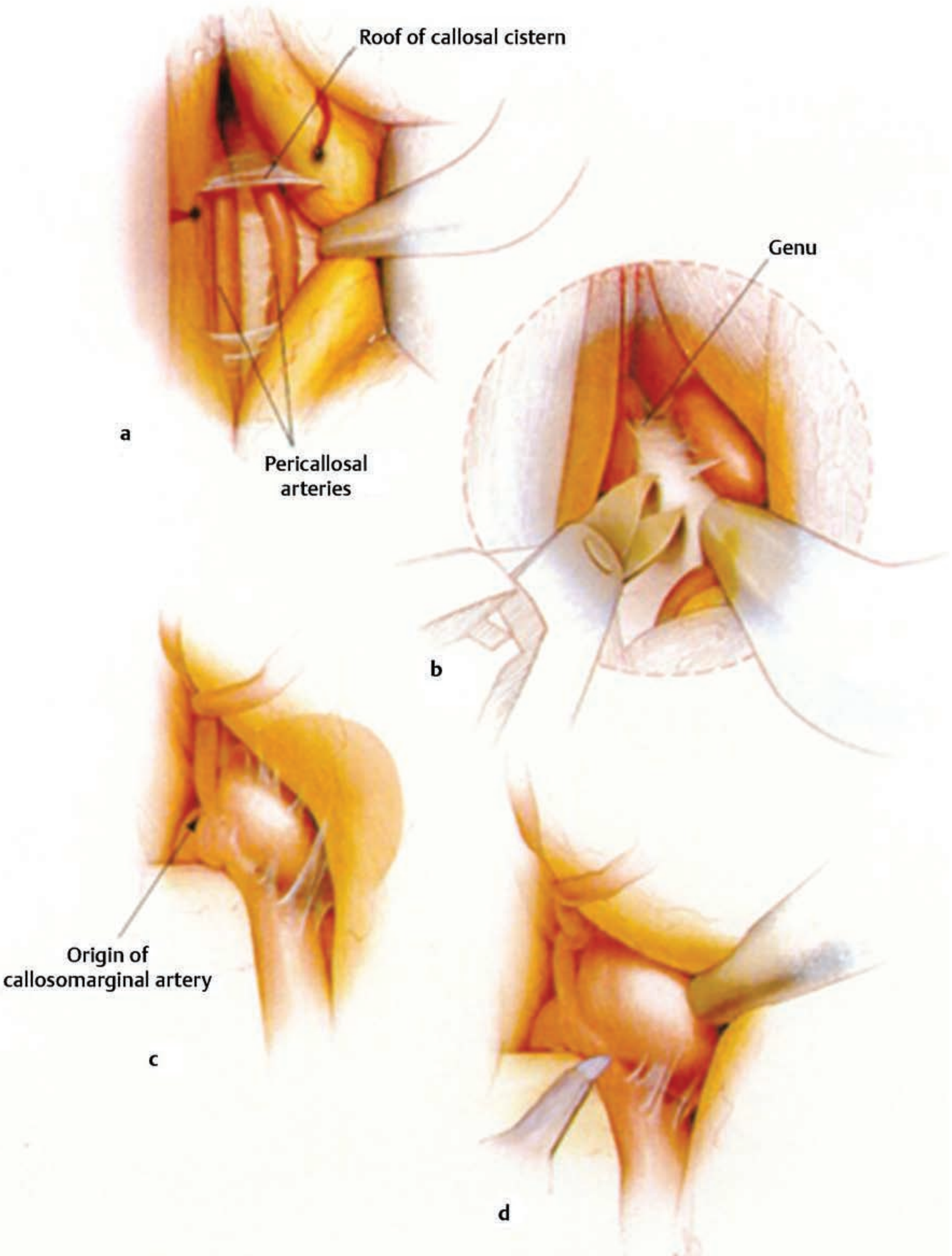

Fig. 10.5 Identification of the transverse arachnoidal bands of the cistern of the corpus callosum marks the initial entrance into the subarachnoid space **(a)**. The efferent pericallosal vessels will be seen within this cistern. Sharp microdissection from distal to proximal along both pericallosal arteries should be confined to the inferior (ventral) aspect of the vessel because the aneurysm and callosomarginal origins are usually located superiorly (dorsally). The pericallosal arteries will be seen to dive inferiorly at the genu of the corpus callosum **(b)**. Proximal control is gained once the pericallosal artery proximal to the callosomarginal artery is identified. Dissection from proximal to distal along the anterior surface of the anterior cerebral artery will disclose the origin of the callosomarginal artery. This vessel defines the proximal neck of the aneurysm and should be carefully mobilized off the aneurysm **(c)**. The distal neck is then defined from the efferent pericallosal artery and explored to ensure that no vessels are in the potential path of the clip blade **(d)**.

very low anterior trajectory is taken, proximal arterial control is achieved later with the interhemispheric approach than with traditional skull base approaches.[12] Thus, extreme care must be taken not to overly manipulate adherent brain, clot, or distal arteries during early microsurgical exposure.

If a frontal lobe hematoma limits further exposure, a small cortical incision may be made to partially decompress the brain. Perianeurysmal clot should be left in place so as not to destabilize the rupture site. The goal of the dissection at this point is to obtain proximal arterial control with as little manipulation of the aneurysm as possible. This is accomplished by expanding the exposure so that the course of the right and left pericallosal vessels can be seen simultaneously. The aneurysm usually points superiorly and forward along with the callosomarginal artery, so it can be helpful to dissect forward on the ventral surface of the pericallosal vessels to where they begin to dive around the genu of the corpus callosum (▶ Fig. 10.5b). Resecting a small portion of the corpus callosum genu is an option that often brings the proximal pericallosal trunks into view, allowing the parent artery to be prepared for temporary occlusion.[7]

The aneurysm can now be freed from the adherent clot, brain, and pericallosal artery. DACA aneurysms invariably arise distal to the origin of the callosomarginal artery which is typically the first noted branch from the dorsal pericallosal artery wall during this approach (▶ Fig. 10.5c). The callosomarginal artery–proximal aneurysm neck interface is delineated. Once this is accomplished, the pericallosal artery–distal aneurysm neck interface is developed (▶ Fig. 10.5d). The contralateral pericallosal artery may be densely adherent to the aneurysm and may need to be mobilized from the aneurysm neck region by tedious sharp dissection.

The clipping strategy will be unique in each case and dependent on the exact characteristics (geometry, calcification, thrombosis, etc.) of the aneurysm at hand. Clipping parallel to the parent artery with as few clips as necessary is ideal. Creative clipping strategies utilizing fenestrated clips, perpendicular clipping, multiple stacked clips, or intersecting clips may be necessary to occlude the aneurysm and reconstruct the parent artery. In most instances, brief episodes of temporary trapping are well tolerated in this location due to robust collateralization. This can allow for elective deflation of the aneurysm prior to final dissection and definitive clipping. We routinely utilize micro-Doppler ultrasound and intraoperative catheter angiography to confirm satisfactory clipping. Reactive vasospasm due to temporary clipping or artery manipulation is treated by topical application of papaverine-soaked Gelfoam (Pfizer Inc., New York, NY).

10.3.5 Closure

The dura is closed in a watertight fashion using interrupted or running 4–0 silk sutures. The bone flap is secured over a dural substitute with titanium miniplates and screws. The galea is closed as a separate layer with braided, absorbable sutures and the skin is stapled.

10.4 Postoperative Management Including Possible Complications

As with all operations aimed at microsurgical aneurysm repair, parent or efferent artery injury or occlusion represents the greatest source of complications. Embolization from the aneurysm dome and tearing of the thin-walled aneurysm neck–artery interface have both been reported.[13] Prolonged trapping can lead to ischemia in the ACA territory. In addition to these general risks, the degree of brain retraction necessary to treat distal anterior cerebral aneurysms is not insignificant, and the risk to frontal bridging veins is real with interhemispheric approaches. Every effort should be made to preserve these veins. Venous infarction may present in a delayed fashion, so a high degree of suspicion should be maintained for several days postoperatively. Angiographic vasospasm and delayed cerebral ischemia remain leading causes of morbidity following SAH. Fortunately, patients with DACA aneurysms do not usually have diffuse thick clot in the basal cisterns. However, although life-threatening angiographic vasospasm and delayed cerebral ischemia may be less common, focal distal spasm involving the distal ACA segments may be difficult to treat with angioplasty and hemodynamic therapy alone. We have found intra-arterial calcium channel antagonists delivered through microcatheters to be very helpful in managing this situation.

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11 Pterional Transsylvian and Extended Approaches for Upper Basilar Aneurysms

Babu G. Welch and H. Hunt Batjer

Abstract

The cerebrovascular specialist is responsible for the judicious application of both endovascular and surgical techniques to achieve durable treatment of an aneurysm in any location. This is particularly true in lesions that arise from the upper basilar artery. This chapter considers the upper basilar to include aneurysms and potentially other lesions that arise from the basilar bifurcation, proximal posterior cerebral (P1 segment of the posterior cerebral artery [PCA]), or superior cerebellar arteries (SCA). While the basilar apex aneurysm has largely been relegated to endovascular therapy, it is important to understand that more lateral and inferior aneurysm locations (PCA and SCA aneurysms) have anatomical characteristics that frequently challenge endovascular techniques. These characteristics include variable native vessel diameters that create abrupt transitions from a larger to a smaller diameter. This chapter reviews the surgical anatomy of the upper basilar artery as well as anomalies that may challenge surgical decision-making and procedural execution. Our discussion of the surgical anatomy will occur with the understanding that, in the endovascular era, a limited number of neurosurgeons will become comfortable with the surgical management of aneurysms of the upper basilar region. We urge the use of the pterional transsylvian approach to most lesions of the upper basilar artery since this approach is also the "workhorse" that provides access to the anterior circulation aneurysm and the comfort that comes with a familiar approach to an unfamiliar location cannot be underestimated.

Keywords: cerebral aneurysm, basilar artery, subarachnoid hemorrhage, endovascular treatment

11.1 Anatomy

The basilar bifurcation is in the interpeduncular cistern. This location is bounded by the clivus and posterior clinoid processes anteriorly, the cerebral peduncles posteriorly, the mammillary bodies and posterior perforated substance superiorly, and the medial temporal lobes laterally. On average, the bifurcation is 15 mm posterior to the carotid arteries. The precommunicating (P1) segments of the posterior cerebral artery (PCA) are the branches of the bifurcation that transition to the P2 segments beyond the junction with the posterior communicating arteries. A large posterior communicating artery when associated with a small, or absent, P1 segment is referred to as fetal configuration. Anterior thalamoperforating arteries can arise in abundance from the posterior communicating artery and may factor in to the side of the surgical approach. The oculomotor nerve will religiously follow a course between the PCA and superior cerebellar arteries (SCAs); thus, it is an indispensable landmark when surgical orientation is in question. The SCAs are single or duplicated structures that exit below the oculomotor nerve and will continue below the tentorium; the trochlear nerve will typically enter the tentorial edge anterior to the SCA descent into the posterior fossa. Pontine perforating vessels will become more abundant about 5 mm proximal to the SCA origins; this creates the frequently quoted "perforator-free zone" below the SCAs where proximal basilar clipping can occur for control or sacrifice.

11.2 Preoperative Considerations

In 1989, Batjer and Samson discussed the causes of morbidity and mortality from surgery of aneurysms of the distal basilar artery and appropriately noted that "errors in surgical timing and technical or conceptual errors [can] prove[d] to be of great consequence in patient outcomes."[1] Since that era, significant enhancements in noninvasive imaging have allowed for an enhanced understanding of the anatomy of the basilar apex that contributes to the creation of the surgical concept. When evaluating a surgical approach to the upper basilar, the surgeon should fundamentally understand the following:

- The relationship of the upper basilar to the posterior clinoid process.
- The projection of the aneurysm dome.
- The PCA vessel that is most involved with the aneurysm neck.
- The relationship of the aneurysm dome to the vessels of the basilar quadrification,
- The presence, size, and relationship of the posterior communicating artery to the P1 segments.

These factors, in addition to the hand dominance of the operating surgeon, will frequently determine the side of surgical approach and dictate clip placement. A schematic representation of the basilar apex in a surgical orientation should be clearly understood (▶ Fig. 11.1).

Preoperative investigations for these cases should include computed tomography angiography. This will allow the surgeon to evaluate many of the points mentioned above noninvasively while also revealing the presence of calcification in wall of the aneurysm or related arteries. The relationship of the posterior clinoid process is best evaluated using sagittal reconstructions (▶ Fig. 11.2). In general, the lower the basilar artery bifurcation is in relation to the posterior clinoid processes, the more difficulty the surgeon will have in obtaining proximal control and the more likely that endovascular adjuncts or primary endovascular therapy may be employed. It is also for this reason that extended approaches to the upper basilar aneurysms were initially described.

While the apex of the basilar artery is commonly referred to as a quadrification, most surgeons will observe many variants to this name. Lasjaunias and colleagues provide an excellent evaluation of basilar artery anatomy and its variants.[2] This provides an excellent anatomical foundation for the preoperative

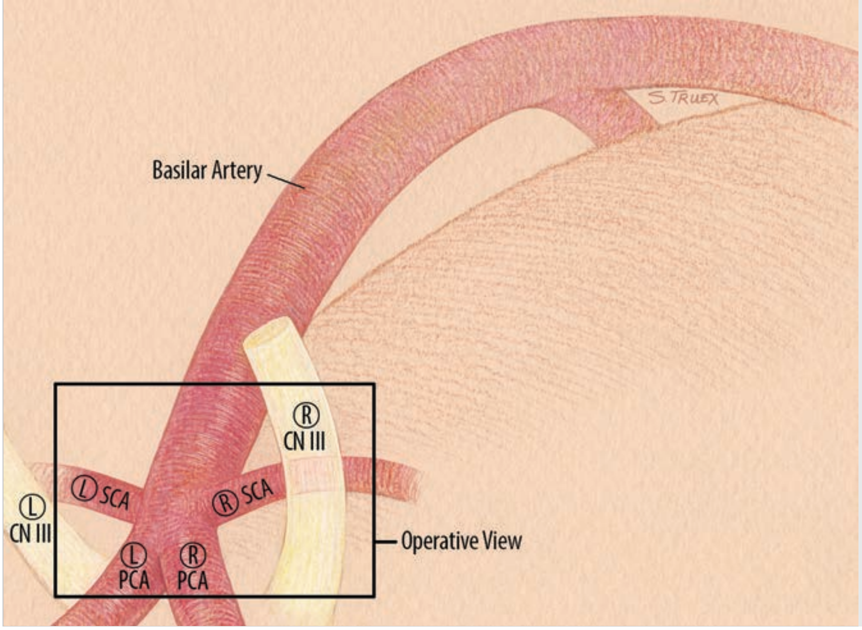

Fig. 11.1 Schematic representation of the basilar quadrification with oculomotor nerve (cranial nerve III) relationship as seen from a right pterional approach.

considerations that are essential to planning a successful transsylvian approach to the aneurysms of the upper basilar artery.

General anesthesia is needed to ensure a brain relatively protected from ischemia by neuroprotective anesthetic drugs, to ensure hemodynamic stability, and to create brain relaxation for the deeper posterior circulation approach. Routine monitoring should include an arterial line, noninvasive blood pressure cuff, five-lead electrocardiogram, pulse oximetry, esophageal stethoscope, temperature probe, Foley catheter, capnograph, peripheral nerve stimulator, and central venous catheter. Pulmonary artery catheters may be used in patients with congestive heart failure or impaired heart function. Where larger lesions may require cardiac pause or rapid ventricular pacing, it may be wise to place defibrillator pads in case of dysrhythmia during cooling.[3,4] Electroencephalography electrodes should be placed for monitoring in case burst suppression is used. Anticonvulsants are not usually administered, perioperative antibiotics are routine, and brain relaxation is facilitated by transcranial placement of a ventricular drain rather than by spinal drainage except in the case of subtemporal approaches.[5] Anesthetic adjuncts for brain relaxation should include mannitol (0.5–1 g/kg) 30 minutes prior to dural opening and hyperventilation to a PaCO$_2$ of 25 to 30 mm Hg. Intraoperative hypothermia to 33 °C may be employed at the discretion of the surgeon.

11.3 The Transsylvian Approach

Wide splitting of the sylvian fissure is a key step during the approach to the upper basilar. The success of this approach in cerebrovascular neurosurgery is dependent on careful tissue mobilization with the goal of circumferential visualization of the aneurysm and optimal illumination of what is ultimately an area that may be up to 12 to 15 mm deep to the carotid cistern. During dissection, the surgeon must exercise extreme care to minimize or create only controlled injury of the arterial, parenchymal, and venous anatomy that is encountered as the surgical corridor narrows. While a thorough understanding of the anatomy of the sylvian fissure makes this possible, we recommend the following goal-oriented approach to understanding sylvian fissure dissection for visualization of the interpeduncular cistern. The surgical goals to achieve in order, where possible, are discussed in the following.

11.3.1 Positioning for Pterional Craniotomy: Stay Away from the Shoulder, so Tilt the Head

Following fixation of the cranial clamp, the head should be rotated to the contralateral side, approximately 45 degrees. This should be followed by neck extension to place the malar eminence at the highest point. Without further modification, this position may cause the temporal lobe to obscure the surgeon's vision and drive him/her toward the ipsilateral shoulder. By slightly tilting the nonsurgical ear toward the ipsilateral shoulder, the surgeon can continue to operate in the sagittal plane and allow a more direct visualization of the upper basilar region.

11.3.2 Craniotomy and Brain Relaxation: Muscle Mobilization and Intraoperative Ventriculostomy

In many cases, the "standard" pterional craniotomy is modified to address the preoperatively evaluated basilar anatomy. For a basilar lesion, we will typically extend the inferior aspect of the incision below the zygoma and perform a subfascial dissection to allow for dissection of the temporalis muscle from the lateral orbit; this will permit posteroinferior retraction of the muscle (▶ Fig. 11.3). In our experience, such muscle mobilization, along with aggressive subtemporal bony removal, obviates the need to routinely consider an orbitozygomatic osteotomy for all but very high and giant lesions. To optimize illumination and minimize brain retraction, emphasis should then be made on

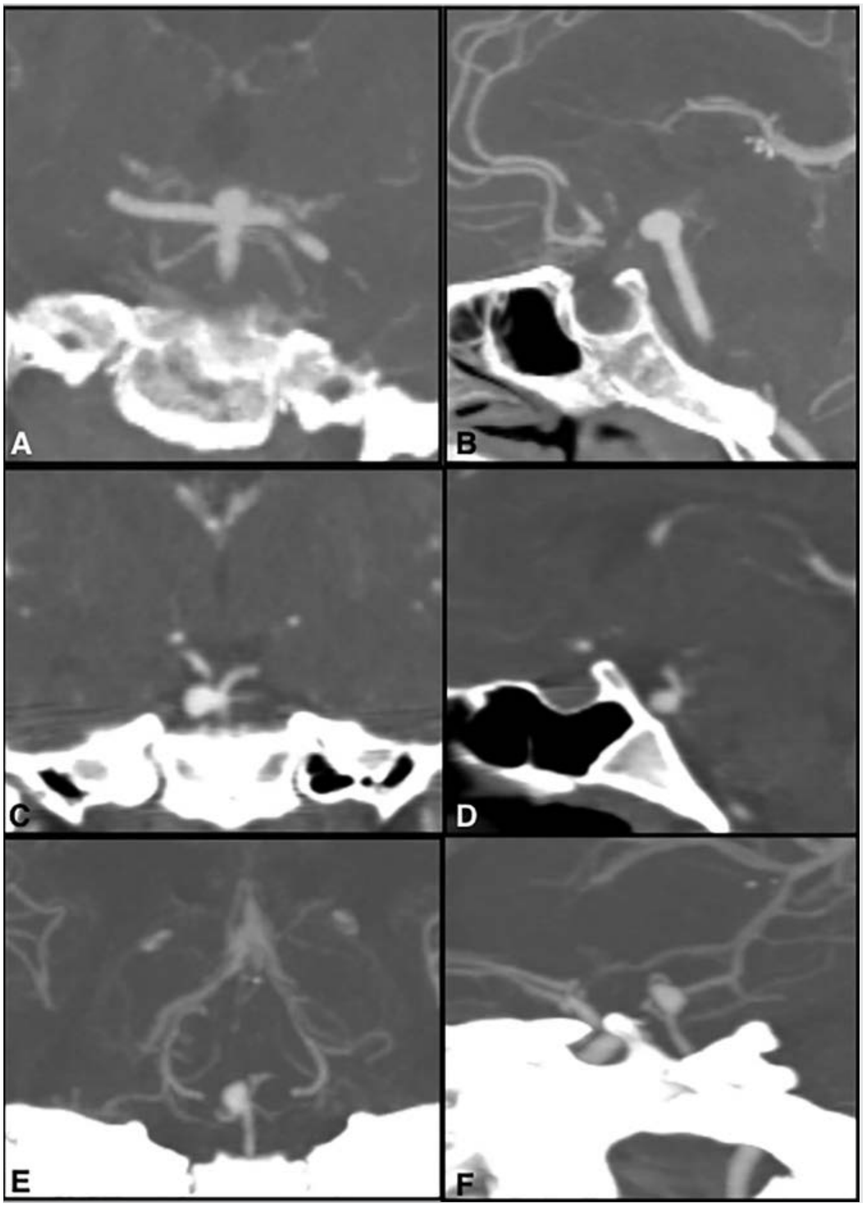

Fig. 11.2 Determining the relationship of the basilar artery to the posterior clinoid process is an essential first step in the surgical plan. This relationship will determine if a pterional (with or without extension) approach is an appropriate choice to the lesions in question. Coronal and sagittal computed tomography angiogram reconstructions are provided to demonstrate high **(a,b)**, low **(c,d),** and level **(e,f)** relationships. The posterior cerebral orientation at the basilar apex is also important to note (e.g., superior orientation should suggest a lower basilar apex location).

generous drilling of the sphenoid wing and placement of an intraoperative ventriculostomy. This technique involves inserting the ventricular catheter perpendicular to the surface of the brain at a point 2.5 cm superior to the lateral orbital roof at the sphenoid ridge and 2.5 cm above the sylvian fissure.[5] This technique should not supplant the anesthetic adjuncts that also maximize brain relaxation.

11.3.3 Sylvian Fissure Dissection: Maximize Arachnoid Dissection/Lobe Retraction, Minimize Venous Injury

The dissection of the sylvian fissure for the purposes of surgery in the interpeduncular cistern should focus on the complete visualization of the middle cerebral artery (MCA) bifurcation to the internal carotid bifurcation. It is equally important to accomplish arachnoid dissection of the anterior cerebral artery, a step that is often dismissed. The goal of maximal dissection is established to allow for both frontal and temporal lobe displacement from the operative field (▶ Fig. 11.4). Suboptimal liberation of the MCA can lead to kinking during clip manipulation, while curtailing subfrontal dissection can lead to obstruction of the surgical view by surgical retractors in the field. We feel it is important to mention the venous injury in the portion of the chapter due to the significant mobilization of the temporal lobe that is essential to this approach. It is during the posterior retraction of the temporal lobe that the surgeon should balance the sacrifice of what may be a network of draining veins to the sphenoparietal sinus with resection of the mesial uncus. Resection of a portion of the uncus is usually well tolerated. Either of these steps will improve visualization but it is likely that controlled uncal resection will be better tolerated than liberal venous sacrifice. The goal of lobe mobilization is to bring the surgeon to the point of manipulation of the oculomotor nerve. The oculomotor nerve should be liberated from arachnoid adhesions along its course to the membrane

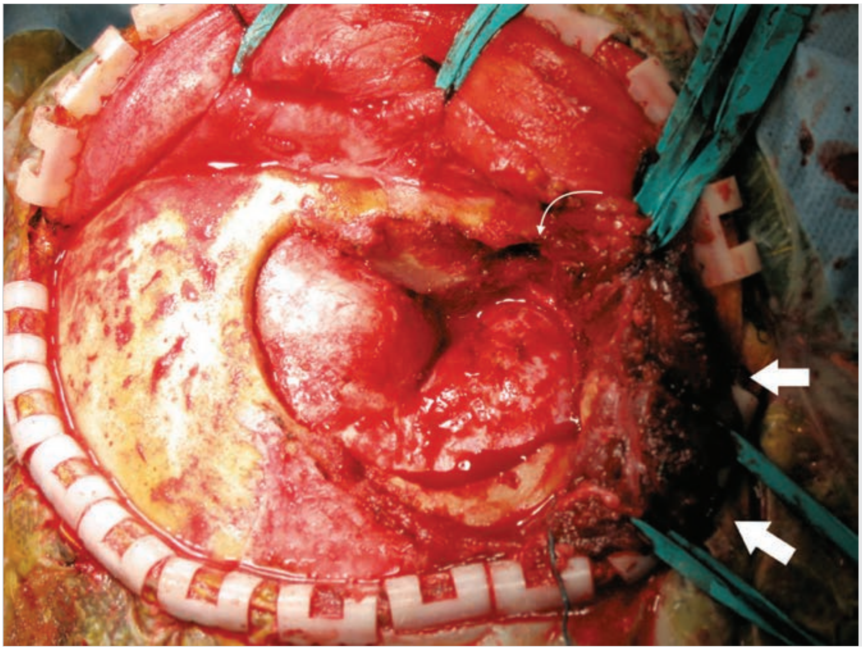

Fig. 11.3 Image of a pterional craniotomy for a basilar apex lesion. *Large arrows* demonstrate the position of the temporalis muscle that is more inferior than many standard pterional approaches. The *curved arrow* demonstrates the improved visualization provided when the lateral orbital rim is exposed by the dissection of the muscle for the inferior temporalis displacement.

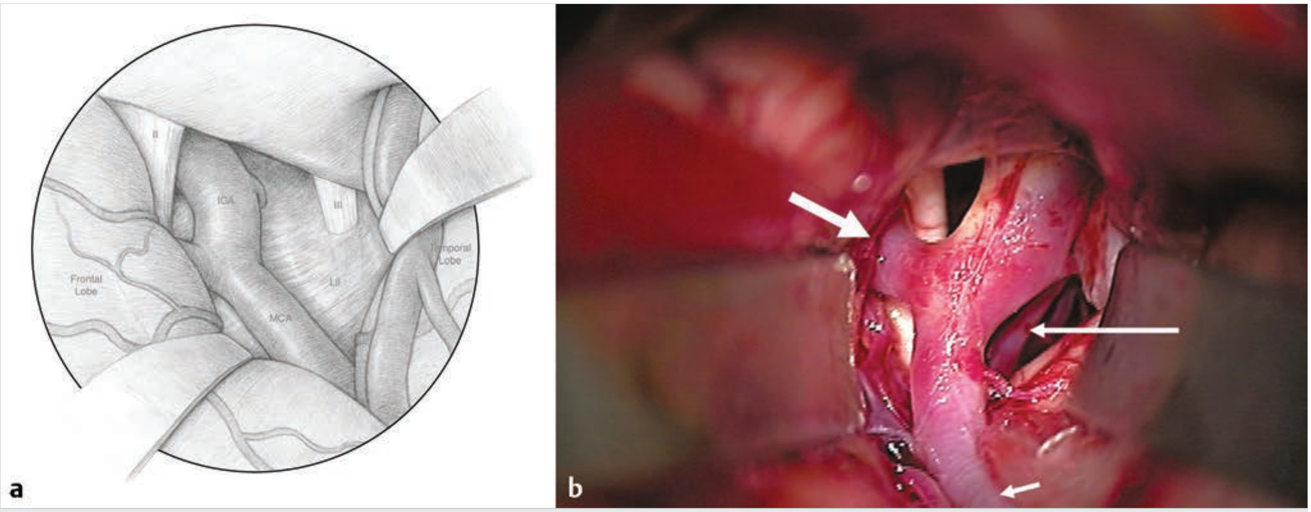

Fig. 11.4 Schematic of the initial exposure of the oculomotor nerve (cranial nerve III) following wide opening of the sylvian fissure **(a)**. The membrane of Liliequist (Lil) should be sharply opened to allow for mobilization of the oculomotor nerve. Dissection of the frontal lobe from the optic nerve (cranial nerve II) will minimize frontal lobe gravitational retraction. Operative view following arachnoid dissection and opening of the membrane of Liliequist **(b)**. *Thick arrow* shows arachnoid dissection along the A1 segment to allow frontal lobe relaxation. *Thin arrow* shows the posterior communicating artery. *Small arrow* shows the middle cerebral artery.

of Liliequist and beyond. As has been mentioned previously, the anatomy of the oculomotor nerve passing between the PCA and SCA is a relationship that the surgeon can depend on.

11.3.4 Visualization of the Basilar Apex: Mobilization of the Oculomotor Nerve and Consideration of the Extended Transsylvian Options

At this point in the procedure, the landmarks of the oculomotor nerve and ipsilateral posterior communicating artery should be well visualized. If a small posterior communicating artery was found to provide minimal contribution to a large ipsilateral PCA, vessel sacrifice with small vascular clips is a useful maneuver to improve visualization of the upper basilar artery. These structures serve as “normal” landmarks that will allow for repeated orientation as surgical dissection of the basilar apex progresses. The mirrored nature of the third cranial nerve facilitates the contralateral dissection that is crucial to complete aneurysm control. Again, the surgeon should keep in mind the relationship of the oculomotor nerve that passes below the ipsilateral PCA and above the most cranial of even a duplicated SCA. This is especially helpful when the surgical field has been altered by a giant aneurysm or the presence of interpeduncular hemorrhage.

Continued arachnoid dissection should reveal the basilar artery and access for proximal control. Retractors may be placed at this point posterior on the gyrus rectus (above the A1) and lateral to the incisura to maximize the sacrifice of the sphenoparietal veins and/or subpial resection of the uncus. Frequently, proximal control in the oculomotor window will inhibit clip placement and at this point the following "extended options" should be considered:

- Further mobilization of the temporal lobe to allow a window lateral to the oculomotor nerve for proximal clip placement; this will move the surgeon lateral (and make the oculomotor nerve the center of surgical field; ▶ Fig. 11.5).
- Opening of the posterior cavernous sinus; this is typically performed following the injection of fibrin sealant via a modified spinal needle without the need for routine sacrifice of trochlear nerve. The maximal opening should occur medial to the first division of the trigeminal nerve

11.3.5 Dome Dissection: What Is the Utility of Temporary Occlusion

The relationship of the aneurysm dome to important thalamoperforating arteries is frequently determined by the projection of dome as well as the size of the aneurysm. While manipulation of the oculomotor nerve may produce a reversible deficit, sacrifice of thalamoperforating arteries is the most devastating complication of basilar apex surgery. The involvement of these vessels is the main distinguishing factor between aneurysms that originate from the PCAs and those originating from the SCAs. The SCA aneurysm rarely involves the thalamoperforating arteries.

The technical aspects of dome dissection rely on fine motor coordination that is a component of adequate exposure and the comfort of the surgeon in this space. A combination of sharp and blunt dissection may be assisted by temporary occlusion to soften the aneurysm and allow for anterior deflection of the posteriorly projecting lesion. Cardiac manipulation using adenosine or ventricular pacing may be useful at this point when a larger lesion prevents circumferential visualization. Caution is required here as the low flow to the perforators produced by these maneuvers makes them difficult to distinguish from arachnoid bands. The low blood flow to the perforators makes them difficult to distinguish from arachnoid bands. The goal of the dome dissection should be to create a safe clip corridor and nothing more.

11.3.6 Decide on Clip Placement

Once the corridor for the clip is created, the size and number of clips can be well approximated using the microdissection instruments. Temporary clip placement should be considered in order to soften the aneurysm and allow more mobilization of it anteriorly to visualize the posterior perforators. Using a corridor lateral to the oculomotor nerve will allow more space in the operative corridor for clip placement; this will require more retraction of the temporal lobe or opening of the posterior cavernous sinus. Burst suppression should be induced and the blood pressure maintained at an adequate level. Cardiac manipulation via adenosine pause or induced ventricular tachycardia can be used in lieu of clip placement to achieve the same effect as temporary occlusion.[3,4] Such techniques obviate the need for additional clips in the small operative space but, at times, this benefit is offset by the unpredictable time that the cardiac effect can be maintained. Few reports suggest that more than 60 seconds of effect can be achieved.

A straight or bayonetted clip blade should be placed across the aneurysm neck with care to cross the neck and not further so as to avoid compromise of the contralateral P1 and its perforators. Alternative clip options include placement of the ipsilateral P1 into a fenestrated clip (▶ Fig. 11.6) or sacrifice of the P1 in the clip to take advantage of an existing and sizeable posterior communicating artery to fill beyond the P2 segment. Both alternatives create a steeper caudal-to-cranial angle for a larger aneurysm (▶ Fig. 11.7, ▶ Fig. 11.8, ▶ Fig. 11.9). When these alternative clip placements are used, a second straight clip may need to be placed above the

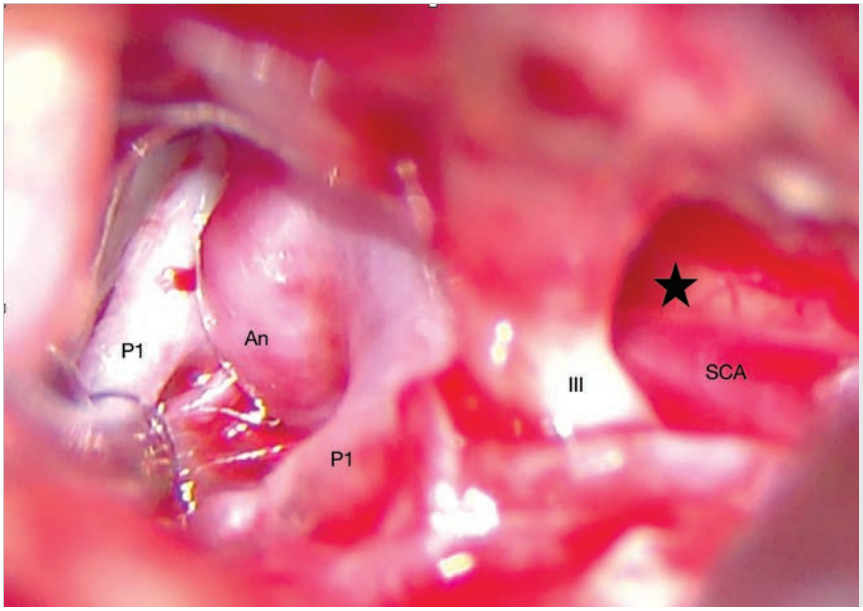

Fig. 11.5 Operative view of a low basilar apex from a right pterional approach following subpial uncal resection. Note the superior projection of the P1 arteries. The oculomotor nerve is passing above the SCA and is more centered in the field to allow for a lateral window to the basilar artery (*star*).

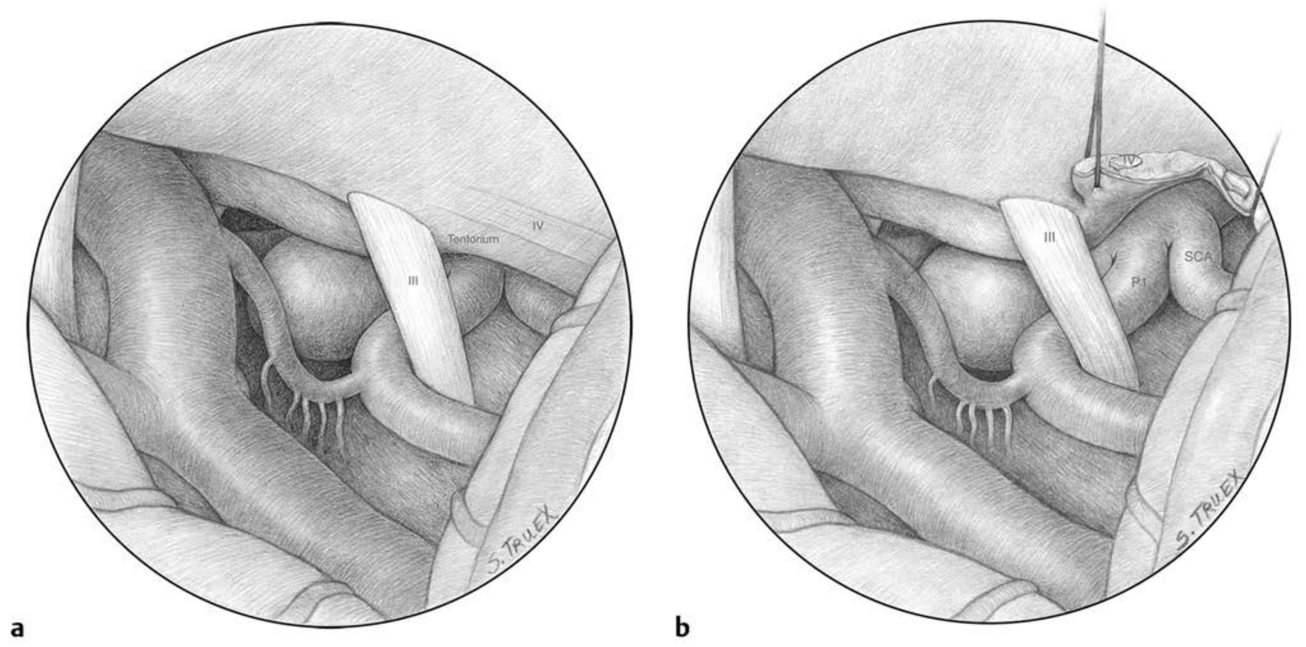

Fig. 11.6 Schematic of a low-lying basilar view before **(a)** and after opening of the tentorium/posterior cavernous sinus **(b)**. Injection of fibrin sealant is a useful adjunct that will decrease cavernous bleeding if a more anterior incision is expected.

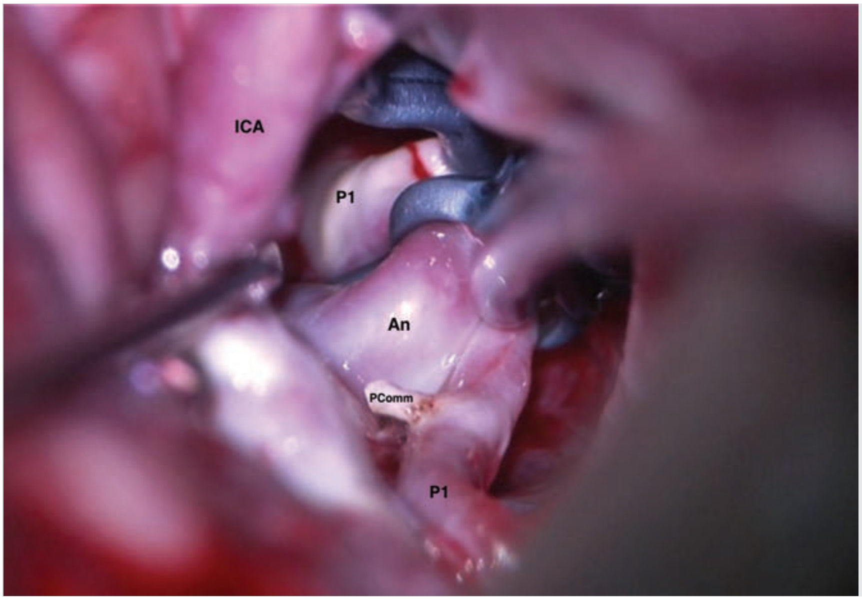

Fig. 11.7 Intraoperative view of right pterional approach to the upper basilar. The posterior communicating artery (PComm) has been sacrificed. The upper clip is providing temporary occlusion when the fenestrated clip is encircling the ipsilateral P1. Residual aneurysm (An) is apparent. This residual aneurysm lobe can be retracted to inspect behind the dome and to then obliterate it with a small straight clip (▶ Fig. 11.9).

ipsilateral P1 to occlude any remaining neck not occluded in the fenestration with the P1. Regardless of technique, the final construct should be inspected to make sure that the aneurysm is obliterated, the P1 segments are patent, and the perforators are free. Rotating the microscope may allow better visualization.

When the aneurysm appears to be clipped, the sac may be punctured with an arachnoid knife. Persistent aneurysm filling may result from the clips not being completely across the neck, the blades being below the contralateral PCA, which was mistaken for the SCA, or the aneurysm wall being too thick or irregular. If there is space, a tandem clip can be placed on the distal or proximal neck or both. The first clip could also be advanced. Once the aneurysm is occluded, the temporary clip on the basilar artery is removed. We assess patency of surrounding arteries using a microvascular Doppler or indocyanine green angiography both before and after clipping. For complex and giant aneurysms, intraoperative angiography is highly recommended.

11.4 Potential Complications

Besides the standard concerns of microvascular dissection, the approach to the upper basilar can create a unique set of complications from the point of uncal resection through creation of the operative window around the oculomotor nerve. If uncal resection is performed, the anterior choroidal artery

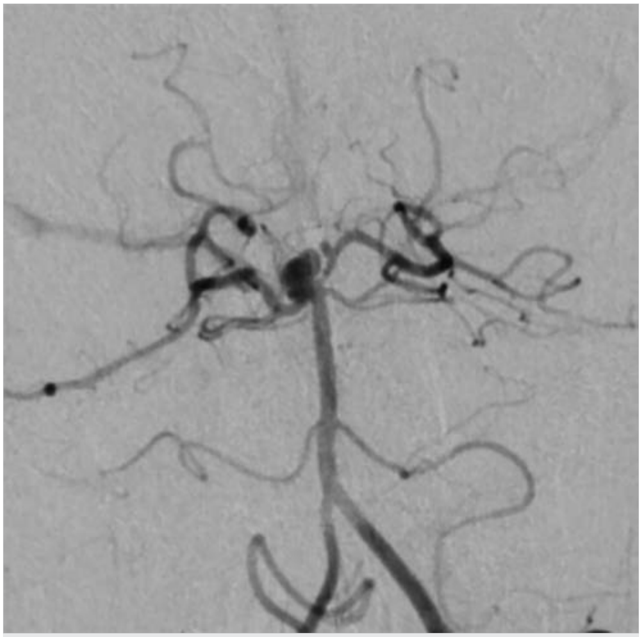

Fig. 11.8 Angiography of a left pterional approach the upper basilar region. In this case, involvement of the P1 origin in the aneurysm and a substantial posterior communicating artery allowed for the P1 to be incorporated into the clip construct. The additional, smaller, P1 aneurysm could also be clipped without concern for P1 patency.

may be injured by violation of the subpial plane. Manipulation of the oculomotor nerve should also be kept to a minimum. Preserving thalamoperforating arteries is arguably the most important and difficult task in this approach that is best achieved through high-magnification dissection during brief periods of temporary occlusion. Intraoperative rupture is a particularly disconcerting event with basilar aneurysms due to the narrow confines and depth of the interpeduncular cistern and the volume of blood flow through the basilar artery. A regimented approach to the identification of the basilar and bilateral P1 vessels will allow for rapid placement of temporary clips should a rupture occur.

11.5 Conclusion

While basilar apex lesions account for more than half of aneurysms in the posterior circulation, this lesion is now rarely treated microsurgically by surgeons who possess exclusively open surgical skill. This not entirely without advantages. The thalamoperforating arteries do not tolerate manipulation well and, despite the increased recurrence that may be observed by endovascular therapy, a closely monitored patient may have many more chances at endovascular reconstruction but only one outcome from a perforator territory infarction.

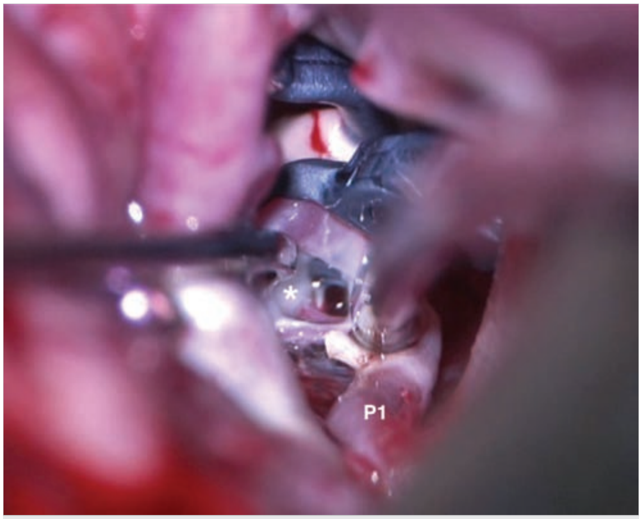

Fig. 11.9 The same view as in ▶ Fig. 11.7. A microdissection tool is used to visualize posterior to the aneurysm (*star*) for perforators involved in the clip placement.

We have made a point in this chapter to comment on the lower perforator density surrounding aneurysms of the proximal PCA and SCA vessels. These lesions have endovascular challenges and may be better approached by direct surgical techniques. Application of the techniques suggested in this chapter will allow for excellent exposure and management of the upper basilar lesions that should continue to be considered for surgical management.

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12 Orbitocranial Zygomatic Approach for Upper Basilar Artery Aneurysms

Antonio Bernardo and Philip E. Stieg

Abstract

Aneurysms arising from the basilar apex are perhaps the most technically challenging aneurysms to treat microsurgically. Endovascular techniques have an important role to play in the treatment of basilar artery aneurysms. However, giant or wide-necked aneurysms and aneurysms that involve the posterior cerebral artery at its neck are not usually amenable to endovascular treatment, and direct microsurgical repair remains the appropriate treatment for patients with these lesions. Conventional approaches used to treat basilar apex aneurysms include the pterional and subtemporal approaches. The anatomy of the basilar artery and the aneurysm and its relationships to bone influence the approach to use. The relationship between the aneurysm and upper clivus (posterior clinoid process) is crucial. Aneurysms located within 5 mm of the dorsum sellae are considered typical. If they occur higher or lower than that point, they are considered high lying or low lying, respectively. High-lying basilar apex aneurysms are partially obscured by the anterior clinoid process and by the anterior petroclinoid ligament. Low-lying aneurysms are covered by the anterior and posterior clinoid processes, the dorsum sellae, and the anterior petroclinoid ligament. The orbitozygomatic osteotomy supplements the exposure achieved by the pterional craniotomy. With this approach, the basilar apex and the four vessels arising at the apex can be identified through the carotico-oculomotor triangle. We routinely use intraoperative indocyanine green videoangiography to verify obliteration of the aneurysm and patency of the posterior cerebral, superior cerebellar, and perforating arteries after clip application. Postoperative care includes neurological intensive care monitoring. A new neurological deficit after surgery is usually investigated with a computed tomography (CT) scan to rule out a hemorrhage or hydrocephalus.

Keywords: basilar apex, basilar artery aneurysm, microneurosurgery, orbitozygomatic osteotomy, anterior clinoid, upper clivus, carotico-oculomotor triangle, clipping, videoangiography

12.1 Introduction

Aneurysms arising from the basilar apex are perhaps the most technically challenging aneurysms to treat microsurgically because the exposure is deep, visibility is poor, and maneuverability is limited. Furthermore, the margin for error is slim because of the rich network of critical perforating arteries in the vicinity that irrigate the brainstem and thalamus and because of the limited proximal and distal control of the aneurysm.

12.2 Patient Selection

Endovascular techniques have an important role to play in the treatment of basilar artery aneurysms, and new methods and devices are almost continuously being brought forward. However, giant or wide-necked aneurysms and aneurysms that involve the posterior cerebral artery at its neck are not usually amenable to endovascular treatment, and direct microsurgical repair remains the appropriate treatment for patients with these lesions (▶ Fig. 12.1a, b).

Conventional approaches used to treat basilar apex aneurysms include the pterional and subtemporal approaches. The

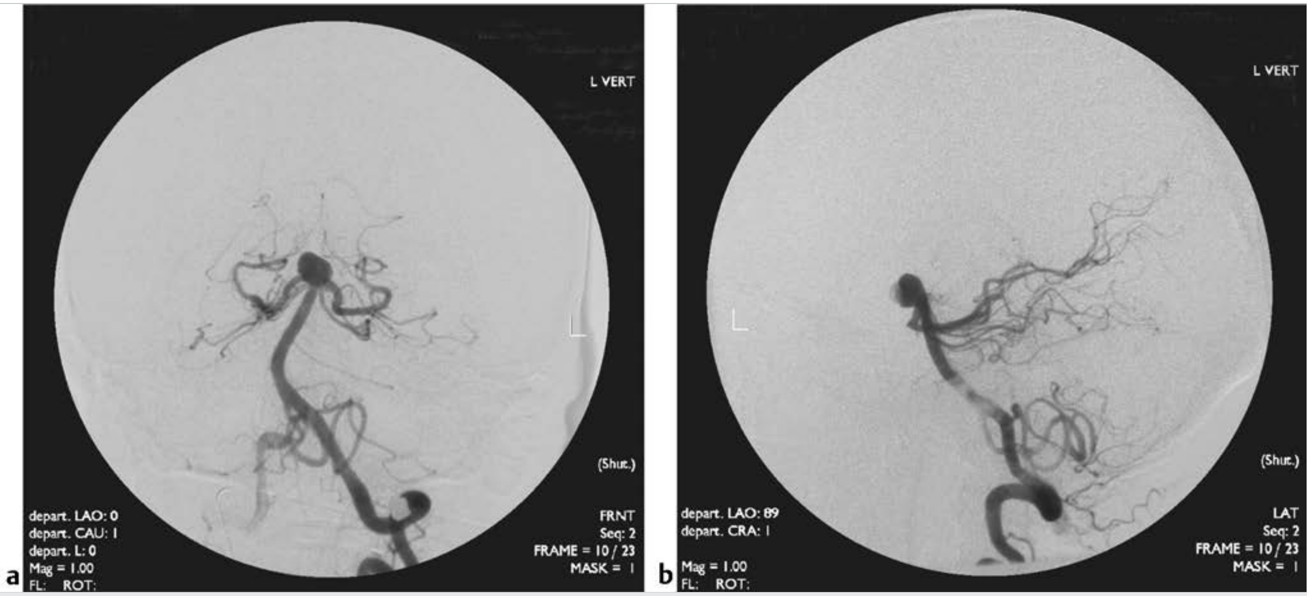

Fig. 12.1 Cerebral angiography. **(a)** Anteroposterior and **(b)** lateral views of a large basilar apex aneurysm.

pterional approach has the advantages of ability to attack the aneurysm from its neck, with a good perspective on the interpeduncular cistern, and the ability to create different surgical corridors. These corridors include the opticocarotid window and the carotid oculomotor window. The window between the precommunicating segment of the anterior cerebral and sphenoidal segment of the middle cerebral artery and optic tract can be used as well. Complexities arise when these lesions are hidden by surrounding anatomical structures. In these instances, conventional approaches often do not provide enough working area.

In the pterional approach, both the subtemporal and the transsylvian routes eventually permit visualization of the clival region through the same deep windows: the carotico-oculomotor window and, occasionally, the opticocarotid window. The ventral brainstem exposure through the pterional approach is limited by the anterior and posterior clinoid process and the petroclinoid folds in the caudal direction and by the orbital rims and zygomatic arch in the rostral direction (▶ Fig. 12.2).

The subtemporal approach offers less arachnoid dissection initially and a better view of the posterior aspect of the aneurysm and any associated perforating arteries. The required temporal lobe retraction is better tolerated when the aneurysm is unruptured and, if ruptured, in cases where acute posthemorrhage hydrocephalus can be treated at the time of craniotomy. However, in obese patients and patients with ruptured aneurysms, the subtemporal approach can be associated with hemorrhagic infarction of the temporal lobe due to retraction and/or damage to the bridging veins.

The anatomy of the basilar artery and the aneurysm and its relationships to bone influence the approach to use. The basilar bifurcation and neck of the aneurysm can be as rostral as the mammillary bodies and the floor of the third ventricle, or there can be a “low” bifurcation below the pontomesencephalic junction. The relationship between the aneurysm and upper clivus (posterior clinoid process) is crucial. Aneurysms located within 5 mm of the dorsum sellae are considered typical. If they occur higher or lower than that point, they are considered high lying or low lying, respectively (▶ Fig. 12.3a, b).

High-lying basilar apex aneurysms are partially obscured by the anterior clinoid process and by the anterior petroclinoid ligament. Low-lying aneurysms are covered by the anterior and posterior clinoid processes, the dorsum sellae, and the anterior petroclinoid ligament (▶ Fig. 12.2). These aneurysms may require skull-base surgical techniques for clipping. The main principle is maximization of bone resection. This allows the surgeon to work within a wide corridor, which facilitates the use of surgical instruments and minimizes retraction of the brain.

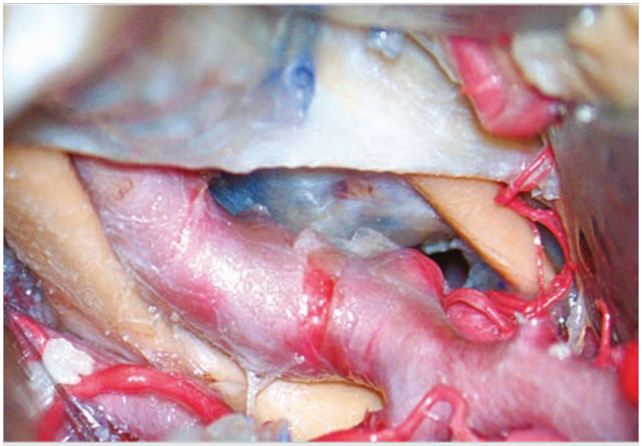

Fig. 12.2 The basilar apex exposure through the surgical corridors of the pterional approach is limited by the anterior and posterior clinoid process and the petroclinoid folds in the caudal direction and by the orbital rims and zygomatic arch in the rostral direction.

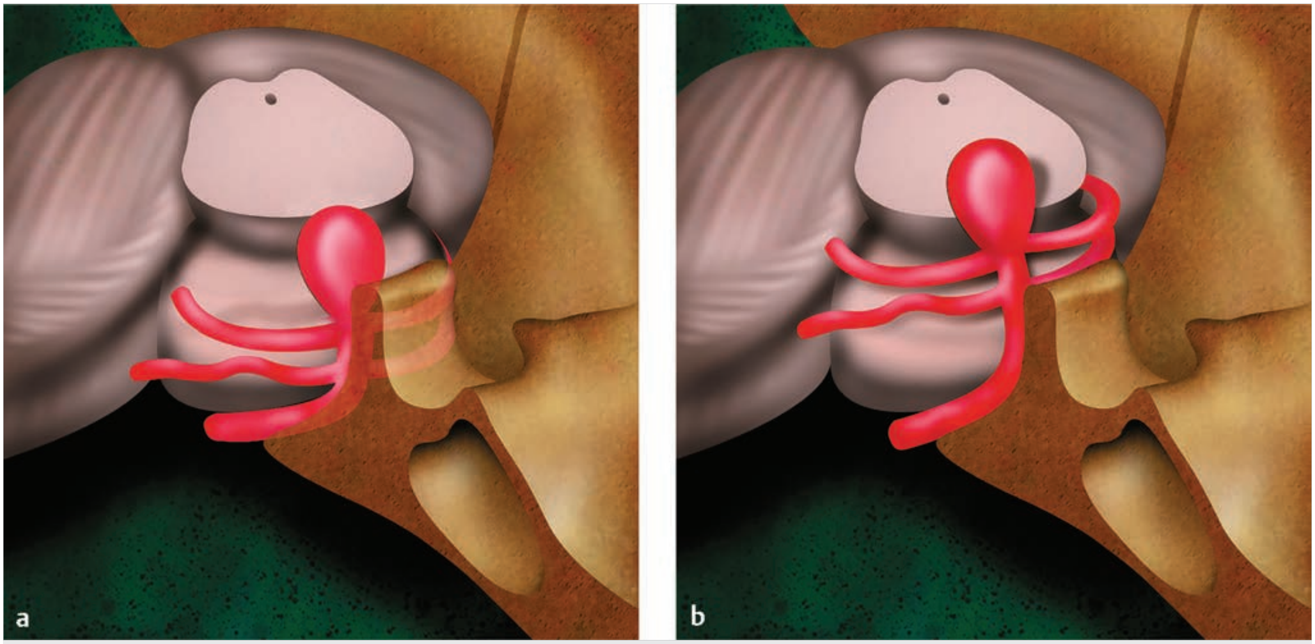

Fig. 12.3 Artist’s depiction of the relationship between the aneurysm and upper clivus. **(a)** Aneurysms located lower than the dorsum sellae are considered low lying. **(b)** Aneurysms that occur higher than the dorsum sellae are considered high lying.

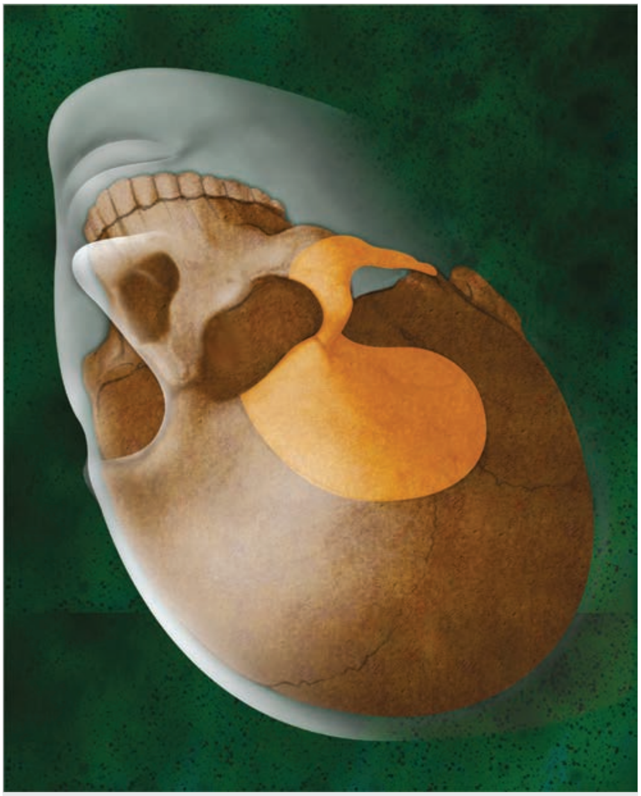

Fig. 12.4 The orbitozygomatic osteotomy consists of removal of the superior orbital rim, orbital roof, and zygoma with the inferior mobilization of the temporalis muscle.

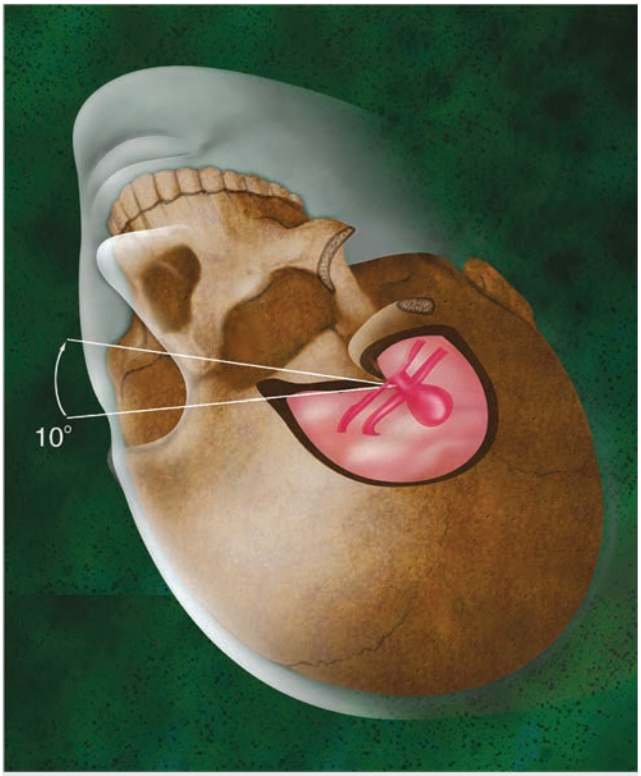

Fig. 12.5 In cases of high-riding lesions, the orbitozygomatic craniotomy increases the angle (10 degrees) over which this region can be viewed by removal of the superior orbital rim and the orbital roof. This technique offers a wide angle of exposure of the basilar apex, reduces the need for brain retraction, and gives a direct line of view to the undersurface of the medial frontal lobe.

The orbitozygomatic osteotomy supplements the exposure achieved by the pterional craniotomy. It consists of removal of the superior orbital rim, orbital roof, and zygoma with the inferior mobilization of the temporalis muscle (▶ Fig. 12.4). In cases of high-riding lesions, the increased angle (10 degrees) over which this region can be viewed after an orbitozygomatic craniotomy becomes crucial (▶ Fig. 12.5). This technique alone offers a wide angle of exposure of the basilar apex and interpeduncular and prepontine cisterns, reduces the need for brain retraction, and gives a direct line of view to the undersurface of the medial frontal lobe.

The extended orbitozygomatic approach is used to access low-lying basilar apex aneurysms and is based on the standard orbitozygomatic craniotomy, with the addition of resection of the anterior clinoid process and the upper clivus (▶ Fig. 12.6).

12.3 Radiological Evaluation

Catheter angiography has been and remains the gold standard in the preoperative evaluation of patients with basilar artery aneurysms (▶ Fig. 12.1a, b). CT is useful because it provides reconstructed images of the aneurysm with related bony surfaces that can be rotated and viewed from multiple angles. This helps the surgeon to understand the anatomy (▶ Fig. 12.7).

12.4 Preoperative Preparation

Euvolemia, normotension, isotonicity, normoglycemia, and mild hypocapnia are recommended. Profound hypocapnia is not recommended unless indicated for control of brain swelling or surgical exposure. Mild hypothermia (33–35 °C) and barbiturate-induced electroencephalographic burst suppression are also used during temporary clipping, final aneurysm dissection, and permanent clipping. During temporary clipping, avoidance of hypotension and the judicious use of induced hypertension provide additional cerebral protection.

A femoral sheath is placed for intraoperative angiography and electrodes to record the electroencephalographic and somatosensory evoked potential responses. The patient's head is placed in a radiolucent pin head holder, slightly extended, and rotated 10 to 15 degrees to the contralateral side.

12.5 Operative Procedure

A curvilinear incision is planned from just anterior to the ipsilateral tragus, at the level of the inferior border of the zygomatic arch, up to the superior temporal line. The incision then gently curves to terminate at the hairline superior to the contralateral midpupillary line. The posterior limb of the superficial temporal artery should be spared in the event that a microvascular bypass is needed. Only a narrow strip of hair is clipped along the course of the incision. The planned incision site is infiltrated with 10 mL of local anesthetic containing epinephrine. The skin is incised, and hemostasis is obtained with Raney clips. The inferior limb of the incision is completed after the scalp is dissected from the temporalis fascia with a periosteal elevator.

The scalp flap is elevated and the underlying temporalis fascia is exposed. The fascia is sharply incised along the margin of the superior temporal line and elevated separately in a subfascial dissection. This technique protects the frontalis branch of the facial nerve, which lies in the subgaleal fat pad and runs along the superficial surface of this fascial plane. The dissection continues anteriorly to expose the orbital rim, malar eminence, and zygomatic arch. The temporalis muscle is raised separately, exposing the zygomatic root and pterion. The muscle flap is left attached to the cranium at its vascular pedicle in the infratemporal fossa. The skin flap and temporalis muscle are retracted anteriorly and inferiorly with surgical fishhooks attached to a retracting bar.

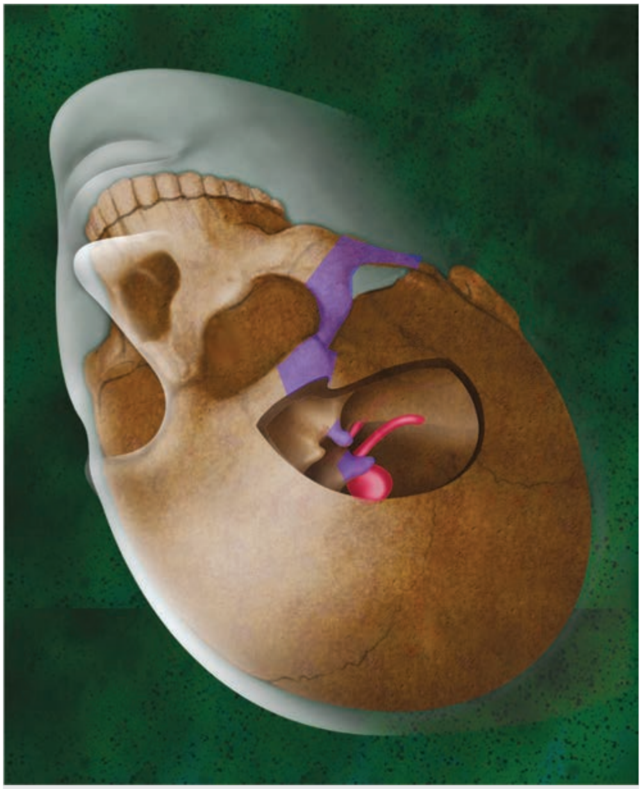

Fig. 12.6 The extended orbitozygomatic approach is based on the standard orbitozygomatic craniotomy, with the addition of resection of the anterior clinoid process and the upper clivus. It is particularly useful to access low-lying basilar apex aneurysms.

A pterional craniotomy is performed and dural tack-up sutures are placed. The periorbita is freed from the lateral and superior aspects of the orbital walls with a Penfield no. 1 dissector, thereby exposing the zygoma and the entire orbital rim (▶ Fig. 12.8). The supraorbital nerve is freed from its bony canal with a small chisel or diamond drill. Orbital and zygomatic osteotomies are completed with a reciprocating saw. The first cut is made across the root of the zygomatic process. The second and third cuts divide the zygomatic bone just above the level of the malar eminence. The fourth cut divides the superior orbital rim and roof. The last two cuts free the lateral orbital wall by connecting the inferior and superior orbital fissures. The inferior orbital fissure is identified by direct vision or by palpating the infratemporal fossa with a no. 4 Penfield dissector. The fifth cut is a short cut made from the inferior orbital fissure to the temporal fossa. The sixth and final cut extends from the lateral margin of the superior orbital fissure to join the fifth cut from the inferior orbital fissure. These bone cuts free the orbitozygomatic bone flap, which is finally elevated (▶ Fig. 12.9a, b). The lateral temporal fossa wall is drilled flush to the middle fossa floor.

In the case of an extended orbitozygomatic approach for low-lying aneurysms, the anterior clinoid process needs to be removed (▶ Fig. 12.10). We prefer to remove the anterior clinoid process extradurally, exposing at the same time the optic nerve in the optic canal. The dura is further elevated from the

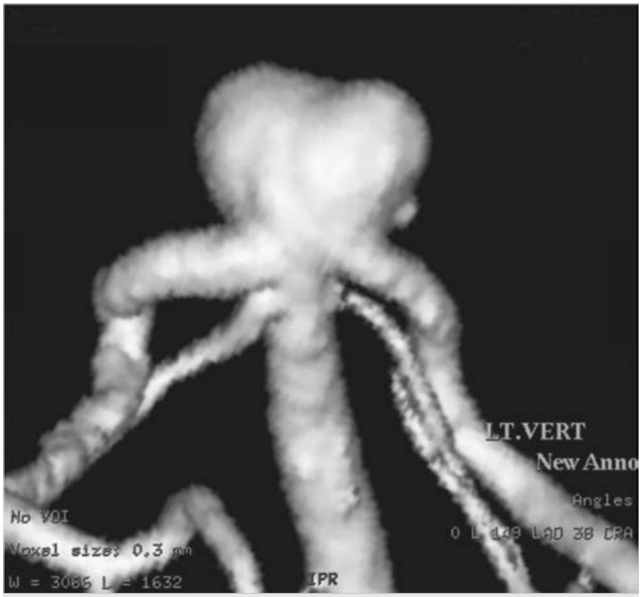

Fig. 12.7 A reconstructed image of a basilar apex aneurysm obtained with computed tomography angiography. The three-dimensional image can be rotated and viewed from multiple angles.

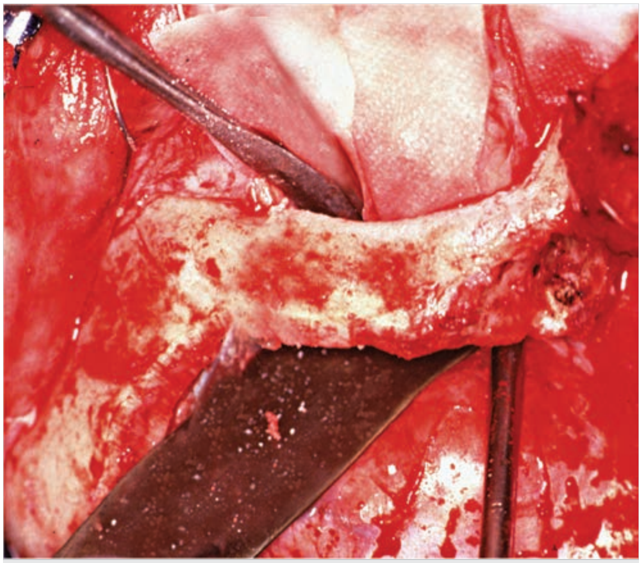

Fig. 12.8 The periorbita is freed gently from the lateral and superior aspects of the orbital walls with a Penfield no. 1 dissector, exposing completely the zygoma and the entire orbital rim.

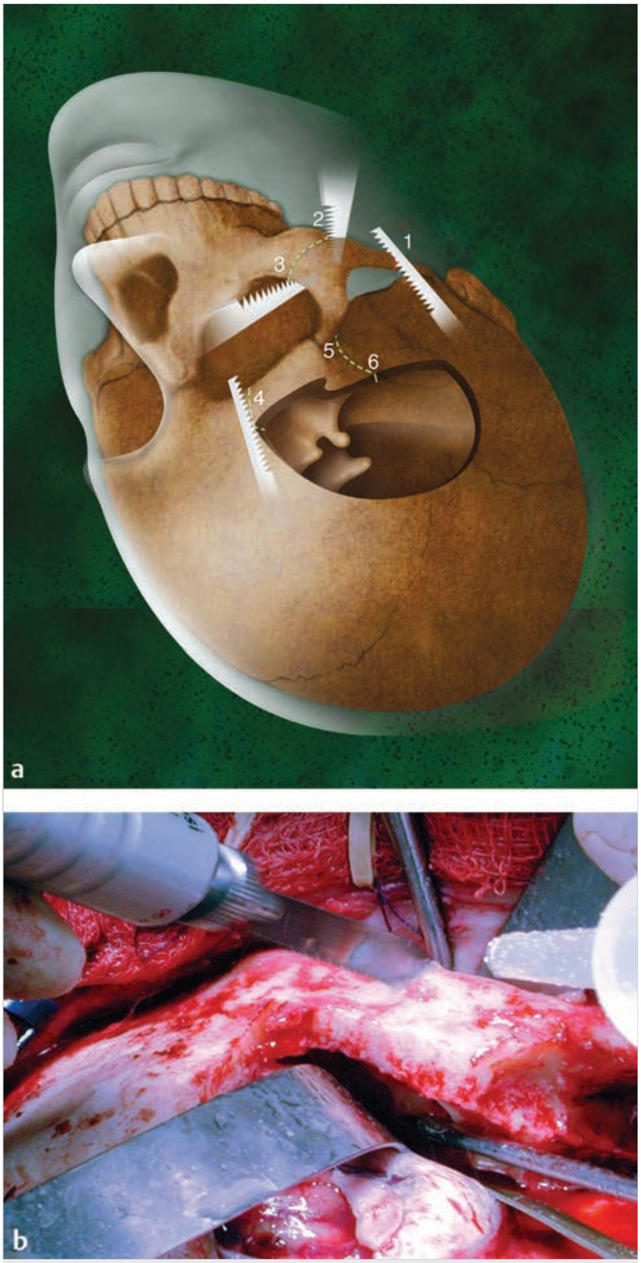

Fig. 12.9 (a, b) Sequential bone cuts are placed across the root of the zygomatic process, the malar eminence, and orbital walls, thereby freeing the orbitozygomatic bone flap, which is finally elevated.

floor of the anterior cranial fossa until the exit point of the optic nerve from its canal is located. The bony roof of the optic canal is thinned with a diamond burr until only a depressible shell of bone remains over the nerve. This shell is fractured and completely removed. At the end of this stage, the optic nerve, free of its bony roof, can be slightly mobilized medially, reducing the likelihood of inadvertent complications during successive steps.

The anterior clinoid process is then removed. The dura lying on the superior and inferior surfaces of the anterior clinoid process is detached from the process, which is then removed with a high-speed drill and diamond burr. During this procedure, bear in mind the surrounding structures: the optic nerve lies medially, the oculomotor nerve covered by the dura is lateral to the process, and the subclinoid segment of the internal carotid artery (ICA) runs inferiorly in the anteromedial triangle.

Once the anterior clinoid process is completely removed, the subclinoid ICA becomes visible. At this point, the optic strut is removed. The dura is peeled from the superior and posterior aspects of the superior orbital fissure and adjacent orbital roof, and with a high-speed drill, the remainder of the lesser sphenoid wing is drilled away. The remainder of the lateral wall of the superior orbital fissure, formed by the greater sphenoid wing, is also removed (▶ Fig. 12.11). This extensive bone removal exposes the optic nerve covered by its dural sheet in the optic canal and the subclinoid ICA, and the proximal and distal dural rings are visible.

A semilunar dural opening is created. The dura is reflected anteriorly and inferiorly over the periorbita and temporalis. In this fashion, the profile of the periorbital contents is flattened, increasing the exposure. The sylvian fissure is then opened microsurgically. The superficial sylvian cistern is entered first, and the dissection proceeds proximally into the opticocarotid cistern. Cerebrospinal fluid is gradually removed from this cistern to increase brain relaxation and minimize the need for brain retraction.

The orbital surface is freed from the optic nerve, and the lamina terminalis is exposed and fenestrated to aspirate more cerebrospinal fluid from the third ventricle for further brain relaxation. The deep sylvian cistern is opened, and the carotico-oculomotor triangle is dissected. Bridging veins between the temporal tip and the sphenoparietal sinus are coagulated and incised. The frontal lobe is elevated, and the temporal lobe is retracted downward and posteriorly. The course of the posterior communicating artery is then visible as it pierces the membrane of Liliequist.

The membrane of Liliequist is incised, and cerebrospinal fluid is further aspirated from the interpeduncular cistern (▶ Fig. 12.12). Once the membrane is opened, the course of the posterior communicating artery can be observed to its junction with the ipsilateral posterior cerebral artery. Sometimes, it is necessary to sacrifice the posterior communicating artery to create more space to ease the manipulation of instruments and the placement of temporary and permanent aneurysm clips. The dissection of the sylvian fissure is completed by skeletonizing the anterior choroidal artery from the ICA to the choroidal fissure.

At this point, the basilar apex and the four vessels arising at the apex can be identified (both posterior cerebral and both superior cerebellar arteries) through the carotico-oculomotor triangle (▶ Fig. 12.13a, b). Rotation of the patient's head increases the visualization of the basilar trunk and contralateral vessels. The basilar artery aneurysm should also be visible. The third cranial nerve between the posterior cerebral and superior cerebellar arteries is identified through this surgical corridor.

For low-lying lesions, the posterior clinoid and upper portion of the clivus can be drilled off with a diamond burr with or without elevating a dural flap (▶ Fig. 12.14a, b, c, d, e). The arachnoid is removed from around the neck of the aneurysm, and both right and left posterior cerebral artery segments are dissected from the aneurysm (▶ Fig. 12.15). An area for

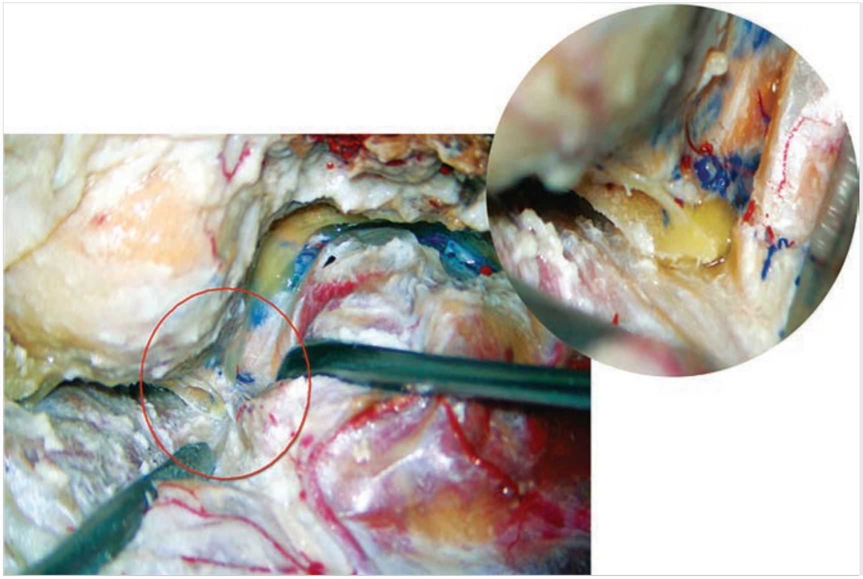

Fig. 12.10 The anterior clinoid process, located between the superior orbital fissure laterally and the optic canal medially, is visible after flattening of the sphenoid ridge.

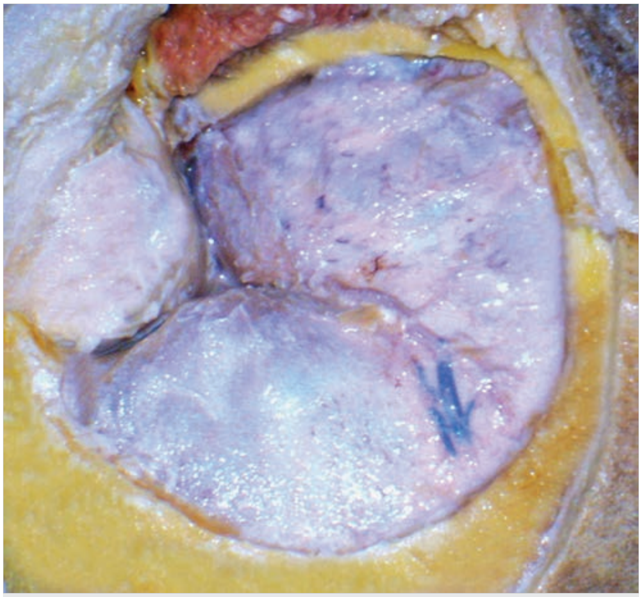

Fig. 12.11 Large parts of the greater sphenoid wing and orbital roof are removed. This extensive bone removal exposes the lateral and superior aspects of the orbit and increases the angle over which structures at the depth of the microsurgical corridor can be viewed.

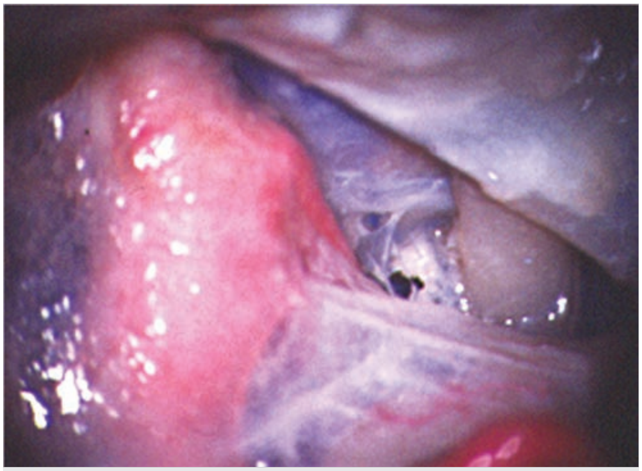

Fig. 12.12 The membrane of Liliequist spans the opening between the carotid artery and the oculomotor nerve and has had a small incision made in it to release cerebrospinal fluid and expose the interpeduncular cistern.

proximal control of the basilar trunk is secured above the posterior clinoid process, and a temporary clip can be placed below the point where the superior cerebellar arteries originate. Usually, this maneuver softens the aneurysm enough for one to proceed with the dissection. Alternatives to temporary clipping are endovascular balloon occlusion and hypothermic circulatory arrest.

Perforating vessels must be sharply dissected free of the aneurysm's neck. All perforators are excluded from the tip blades during final clip placement. Long, straight clips may improve visibility and are guided into place from an inferior trajectory. For large aneurysms, a combination of fenestrated and long, straight clips is preferred. After clip placement, the aneurysm dome can be tilted forward to ensure that all perforators are excluded from the clip blades. At times, a second clip may be needed to free the perforators.

We routinely use intraoperative indocyanine green videoangiography to verify obliteration of the aneurysm and patency of the posterior cerebral, superior cerebellar, and perforating arteries after clip application (▶ Fig. 12.16). Unexpected residual aneurysm or occlusion of a major artery exiting the basilar apex mandates repositioning of the clip or placement of another clip.

At the end of the intradural procedure, the dura is closed in a watertight fashion with a continuous absorbable suture. The orbitozygomatic and cranial bone flap are replaced with titanium microplates and screws. The temporalis muscle and fascia are reapproximated and reattached to the superior fascial cuff on the bone flap.

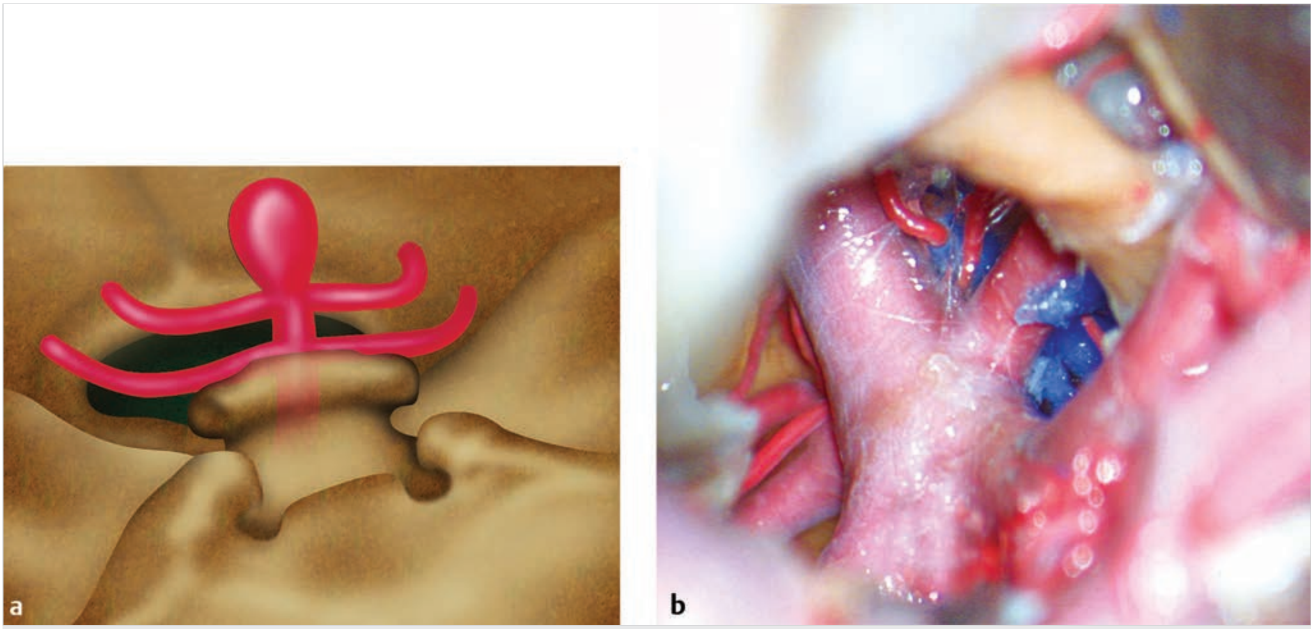

Fig. 12.13 After the membrane of Liliequist has been opened, the basilar apex and the four vessels arising at the apex can be identified (both posterior cerebral and both superior cerebellar arteries) through the carotico-oculomotor triangle. **(a)** Artist's depiction. **(b)** Surgical perspective.

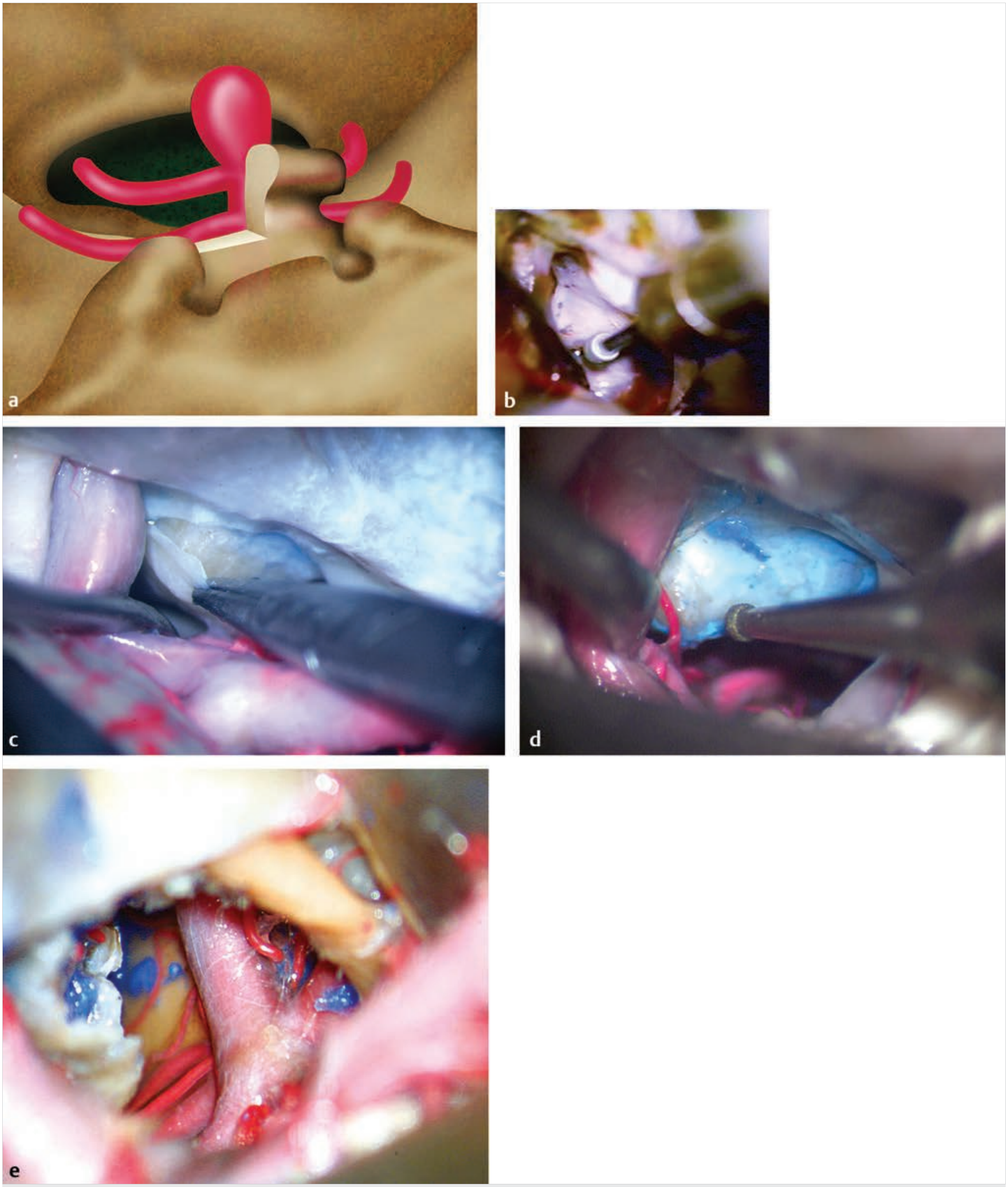

Fig. 12.14 Enhanced exposure of the basilar apex for low-lying lesions. When the basilar apex is more than 5 mm below the posterior clinoid processes in a lateral view, it can be obscured by the posterior clinoid processes and the upper portion of the clivus. **(a)** Artist's depiction. **(b)** In such a case, the posterior clinoid and upper clivus can be drilled away. **(b)** Surgical perspective. This can be done with **(c)** or without **(d)** elevating a dural flap. **(e)** Surgical perspective after removal of the posterior clinoid and upper clivus.

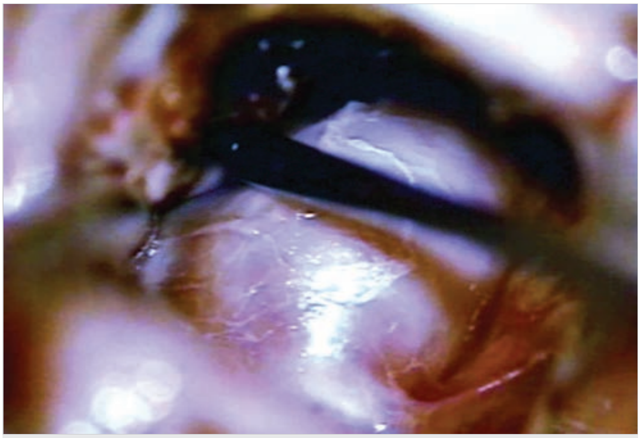

Fig. 12.15 The arachnoid is removed from around the neck of the aneurysm and both right and left P1 segments are dissected from the aneurysm base.

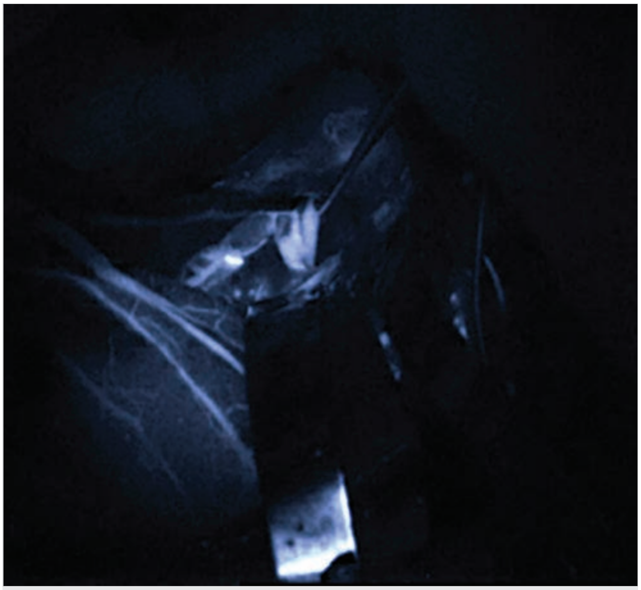

Fig. 12.16 Indocyanine green videoangiography is used to confirm blood flow in parent, branching, and perforating arteries as well as for the assessment of unexpected residual aneurysm after clip application.

12.6 Postoperative Management Including Possible Complications

Postoperative care includes neurological intensive care monitoring. Blood pressure is monitored with an arterial catheter and urine output with an indwelling catheter. Typically, normotensive and euvolemic conditions are maintained. Perioperative antibiotics, glucocorticoids, and anticonvulsant drugs are used variably. After being monitored in the intensive care unit, the patient is transferred to a standard surgical floor, where mobilization occurs. A new neurological deficit after surgery is usually investigated with a CT scan to rule out a hemorrhage or hydrocephalus. Magnetic resonance scanning with diffusion-weighted imaging may be appropriate if an infarction is suspected.

Macdonald, Neurosurgical Operative Atlas: Vascular Neurosurgery, 3rd Ed. (ISBN 978-1-62623-110-8),

13 Subtemporal and Pretemporal Approaches for Basilar and Posterior Cerebral Artery Aneurysms

Feres Chaddad, José Maria De Campos Filho, Mateus Reghin Neto, Axel Perneczky, Gerrit Fischer, and Evandro De Oliveira

Abstract

Depending on the relationship of the basilar bifurcation to the posterior clinoid process, several surgical approaches are used for basilar bifurcation aneurysms, including the subtemporal, temporopolar, pretemporal, pterional, orbitozygomatic, supraorbital, or combined approaches. The subtemporal approach offers a lateral view of the interpeduncular fossa by retracting the temporal lobe superiorly. Advantages over the pterional approach and its variants are that the subtemporal approach provides a better view of the posterior aspect of the basilar bifurcation where critical perforating arteries arise. It also may be easier in cases of anteriorly or posteriorly directed aneurysms. The pretemporal approach combines the advantages of these approaches in one craniotomy.

Keywords: posterior cerebral artery, posterior communicating artery, pretemporal craniotomy, subtemporal approach, vertebral artery

13.1 Patient Selection

Aneurysms at the distal third of the basilar artery including the basilar artery bifurcation and the origin of the superior cerebellar artery from the basilar artery and less commonly aneurysms that originate from the proximal posterior cerebral artery are most commonly repaired by endovascular methods. Nevertheless, in cases of aneurysms with wide necks and/or incorporation of the origins of the posterior cerebral arteries into the aneurysm, open neurosurgical clipping may be the best option.

The surgical approaches to the upper basilar artery include pterional and its extended variations (orbitozygomatic and supraorbital, which facilitate exposure of a high basilar bifurcation), temporopolar, pretemporal, subtemporal, or various combined approaches (▶ Fig. 13.1). Selection of approach depends on several factors including the relationship of the basilar bifurcation to the posterior clinoid processes, direction of orientation of the aneurysm and relationship to the midline, rupture status, presence of additional aneurysms, and size of the posterior communicating arteries. From the pterional direction, aneurysms where the neck is lower than about halfway from the posterior clinoids to the floor of the sella will be difficult to expose; additional exposure usually would be needed to see the basilar artery in order to achieve proximal control.

The pterional approach was popularized by Yasargil.[1,2] It provides a slightly oblique and straight downward view of the anterolateral aspect of the basilar bifurcation that is seen through some combination of the space between the internal carotid artery and optic nerve, between the internal carotid artery and third nerve, above the carotid bifurcation or by opening the tentorium cerebelli lateral to the third nerve.[3,4] Both precommunicating (P1) segments of the posterior cerebral arteries and the origins of the superior cerebellar arteries can be seen. The difficulties are for anteriorly projecting aneurysms where one potentially exposes the dome of the aneurysm first and difficulty seeing the perforators coming off the basilar tip behind the aneurysm.[2,3] The temporopolar approach of Sano is a pterional craniotomy with a more extensive exposure of the temporal lobe.[2,5,6,7,8] The temporal pole is retracted posterolaterally to expose the space between the third nerve and the free edge of the tentorium, creating an anterolateral view of the interpeduncular fossa.

This chapter describes the subtemporal and pretemporal approaches. The pretemporal approach combines the advantages of these approaches in one craniotomy.[9,10,11] This approach is based on extended resection of the sphenoid and temporal bones, wide opening of the basal cisterns, and detachment from the frontal lobe to the temporal lobe. It combines the multiple angles of view offered by the pterional and subtemporal approaches (▶ Fig. 13.2, ▶ Fig. 13.3, ▶ Fig. 13.4, ▶ Fig. 13.5, ▶ Fig. 13.6). The subtemporal approach was first described by Gillingham in 1958 and then popularized by Drake (▶ Fig. 13.7, ▶ Fig. 13.8, ▶ Fig. 13.9). The subtemporal approach is used for basilar bifurcation aneurysms as well as for some aneurysms arising from the superior cerebellar and proximal posterior cerebral arteries. It is most favorable in patients with a low-seated basilar bifurcation in relation to the posterior clinoid process. A prefixed chiasm or low-positioned hypothalamic structures also make the subtemporal approach preferable to others. The true advantage of this approach is the excellent exposure and direct visualization of the back of the aneurysm and thus the vital perforators of the basilar tip and precommunicating (P1) segments of the posterior cerebral arteries. This makes it particularly useful for posteriorly directed aneurysms. When the approach is from the side, the direction of the clip blades tends to be parallel to the basilar bifurcation so there is a low risk of kinking the P1 segments. Limitations include the fact that the contralateral neurovascular structures are less easily seen and controlled. Hence, careful preoperative planning to determine the side of the craniotomy is mandatory. Also, because temporal lobe retraction is required, brain relaxation has to be achievable, so the approach may be more difficult than other approaches in the setting of acute subarachnoid hemorrhage (SAH).

13.2 Anatomy

The posterior cerebral artery originates from the basilar apex immediately distal to the origin of the superior cerebellar arteries and runs into the supratentorial space (▶ Fig. 13.7 and ▶ Fig. 13.8). The P1 segment of the posterior cerebral artery runs laterally from its origin at the basilar bifurcation, in the interpeduncular fossa, until the junction with the posterior communicating artery. The most common branches arising from this segment are the posterior thalamoperforating arteries, the medial posterior choroidal artery, the branch to the quadrigeminal plate, the long and short circumflex arteries, and the

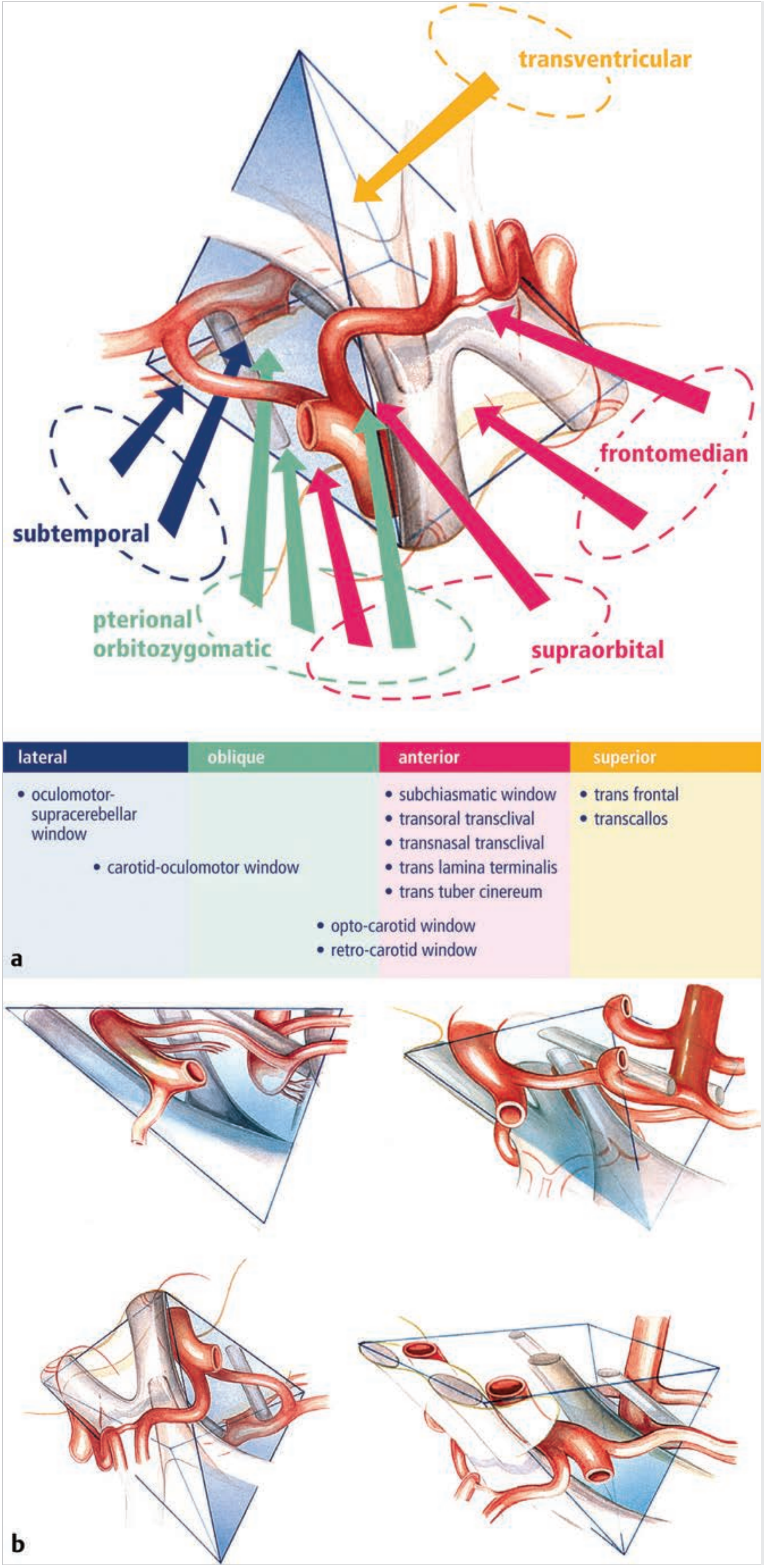
transventricular
frontomedian
subtemporal
pterional
orbitozygomatic
supraorbital
lateral
oblique
anterior
superior
• oculomotor-supracerebellar window
• carotid-oculomotor window
• subchiasmatic window
• transoral transclival
• transnasal transclival
• trans lamina terminalis
• trans tuber cinereum
• opto-carotid window
• retro-carotid window
• trans frontal
• transcallos
a
b

Fig. 13.1 (a, b) A virtual pyramid construction representing the suprasellar structures helps conceptualize the various viewing angles into this space using a subtemporal exposure. The different windows between the anatomical structures can be used through different angles in a more anterior or more posterior approach.

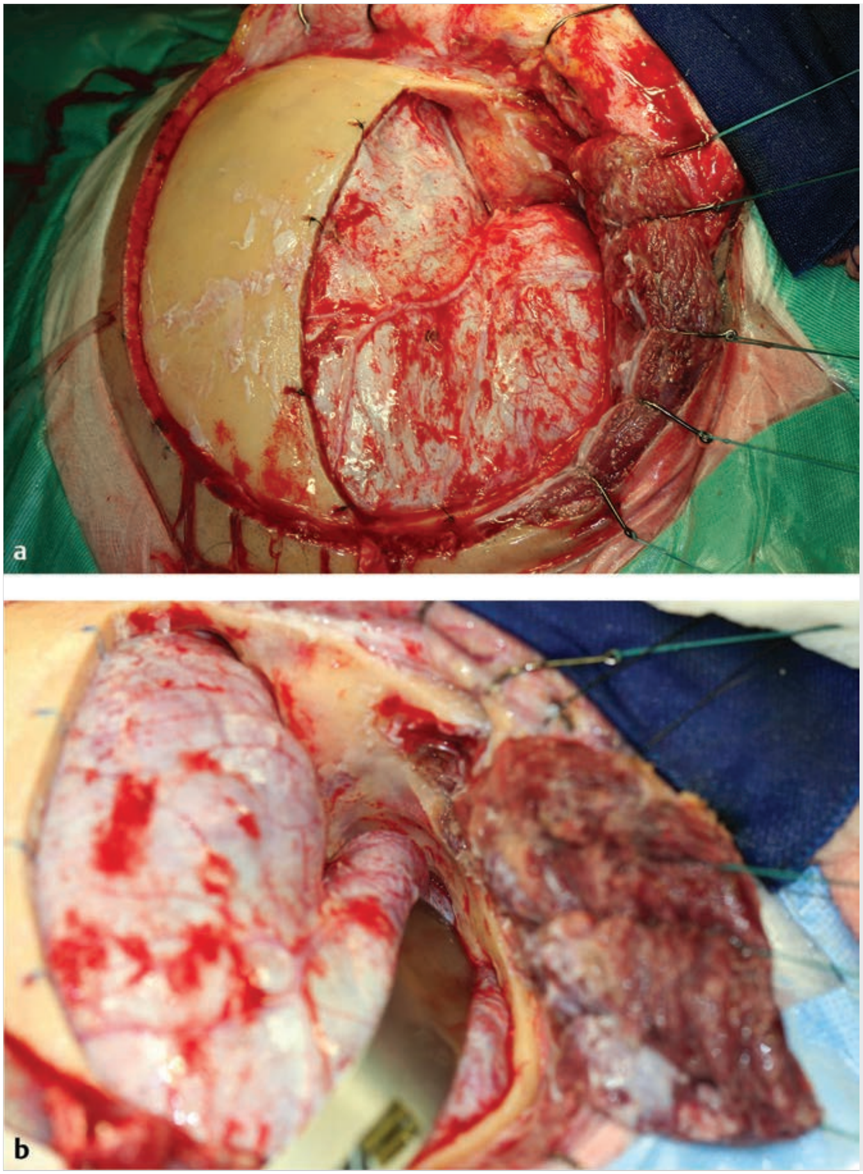

Fig. 13.2 **(a)** Pretemporal craniotomy showing that the greater and lesser wing of the sphenoid, the squamous part of the temporal bone, and the orbital roof are drilled out. **(b)** Pretemporal craniotomy showing subtemporal extradural view.

branch to the cerebral peduncle and mesencephalic tegmentum. The P2 segment begins at the junction with the posterior communicating artery and runs through the crural and ambient cistern until the most posterior and lateral edge of the midbrain. This segment might be divided in two portions: the P2A or crural segment, which courses around the cerebral peduncles, and the P2 P or ambient segment, which courses lateral to the midbrain. Usually, the hippocampal, anterior temporal, and peduncular perforating branches, and, sometimes, the medial posterior choroidal artery, originate from the P2A segment, whereas middle temporal, posterior temporal, common temporal, thalamogeniculate, and lateral posterior choroidal arteries most frequently arise from P2 P.

13.3 Preoperative Preparation

A cranial computed tomography scan with 3D reconstruction is needed to evaluate the direction of projection of the aneurysm, relationship to the posterior clinoid processes, size of the ventricles, and any cerebral atrophy that will give an indication as to how much brain relaxation will be achieved by cerebrospinal fluid drainage. Calcification in the adjacent arteries and aneurysm can be looked for. A magnetic resonance imaging study will show the topographic relationship of the aneurysm to the surrounding structures. A prefixed chiasm or a low-situated hypothalamus can be detected, making the subtemporal approach relatively more favorable. Catheter digital subtraction angiography will demonstrate perforating arteries. An unsubtracted lateral view should be done to determine the relationship of the aneurysm to the posterior clinoid processes.

For basilar artery aneurysm surgery, a full selection of clips, especially fenestrated and bayonetted, is mandatory. Temporary occlusion of the basilar artery may be necessary and can be accomplished by temporary clipping or with endovascular balloon occlusion with a catheter in the basilar artery. Intraoperative angiography is also beneficial to demonstrate the pre- and postclipping situation immediately.

Anticonvulsants are not indicated unless the patient has a seizure before surgery or in the rare case of a patient with epilepsy.

Single injections of perioperative antibiotics are given during surgery. We avoid lumbar drainage and use a drain inserted into the temporal horn of the lateral ventricle at surgery if needed for relaxation. The monitoring consists of evoked potentials for aneurysms that project posteriorly into the pons or mesencephalon.

13.4 Subtemporal Approach

13.4.1 Positioning of the Patient

The patient is positioned supine with the head elevated above the heart level to facilitate venous drainage (▶ Fig. 13.9). The ipsilateral shoulder should be elevated with a cushion to prevent extensive head and neck rotation that could compress the contralateral jugular vein or carotid artery. The head is fixed in a pin head holder, radiolucent if intraoperative angiography is planned, and rotated 80 to 100 degrees to the contralateral side. The zygomatic arch should be in an almost horizontal position. The next important step is to laterally flex the head 15 to 20 degrees to the contralateral side (toward the floor) to provide an ergonomic working position for the surgeon by compensating for the upward angle of the floor of the middle cranial fossa. This maneuver is also important because it promotes gravity-related as opposed to physical retraction of the temporal lobe. Extension of the head (retroflexion) of about 10 degrees may prevent compression of the larynx and the ventilation tube.

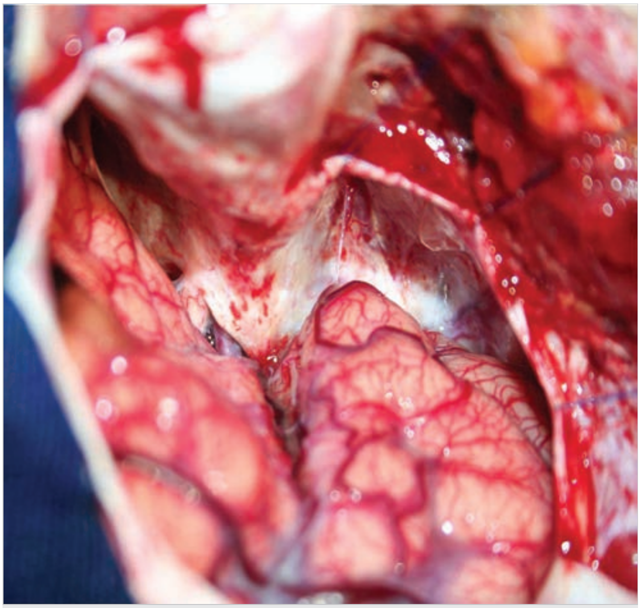

Fig. 13.3 Pretemporal craniotomy exposure allows for microsurgical transsylvian, temporopolar, and subtemporal approaches.

13.4.2 Skin Incision

An epifascial skin incision of around 50 mm is performed in a vertical line from the zygomatic arch superiorly and 10 mm anterior to the external auditory meatus (▶ Fig. 13.10). The subcutaneous tissue is dissected, preserving the superficial temporal artery and auriculotemporal nerve. The fascia of the temporal muscle is incised in a **Y**-shaped fashion (▶ Fig. 13.11, ▶ Fig. 13.12, ▶ Fig. 13.13). The basal leaflet is retracted downward over the zygomatic arch to protect the temporal branch of the facial nerve while the remaining leaflets are retracted bilaterally to expose the temporalis muscle. In some cases, the inferior margin of the muscle can be mobilized bluntly upward to uncover the squamous portion of the temporal bone. In patients with thick temporal muscle, a small vertical incision at the posterior margin is necessary. Postoperative problems with mastication, mandible opening, and muscular atrophy can be prevented and the cosmetic outcome improved by minimizing the exposure and muscle dissection.

13.4.3 Craniotomy and Clipping

The craniotomy is started with a burr hole just above the anterior third of the zygomatic pedicle (▶ Fig. 13.14). Then a straight line parallel to the zygomatic arch is sawed with the craniotome, after

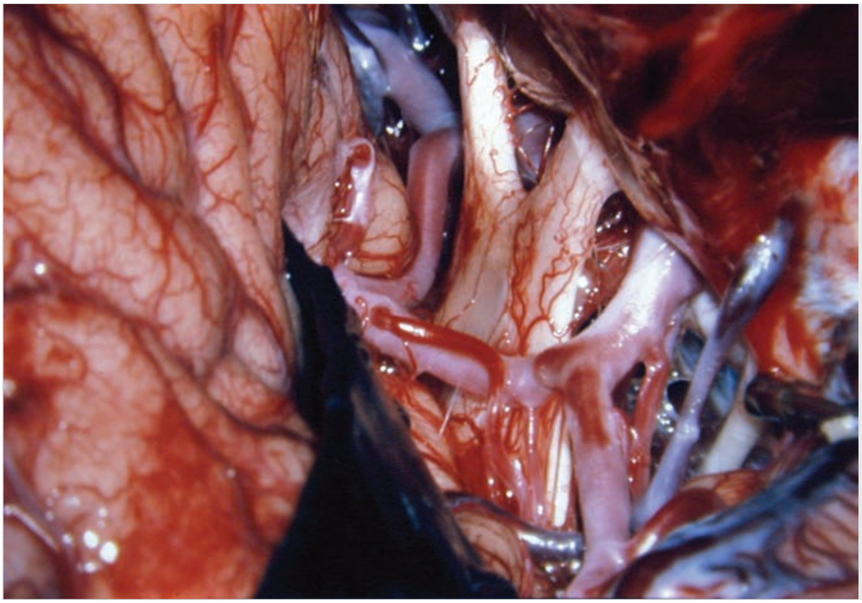

Fig. 13.4 The pretemporal approach allows a large exposure of the basal cisterns and the visualization of the carotid artery and its bifurcation, the optic nerve, and the third nerve.

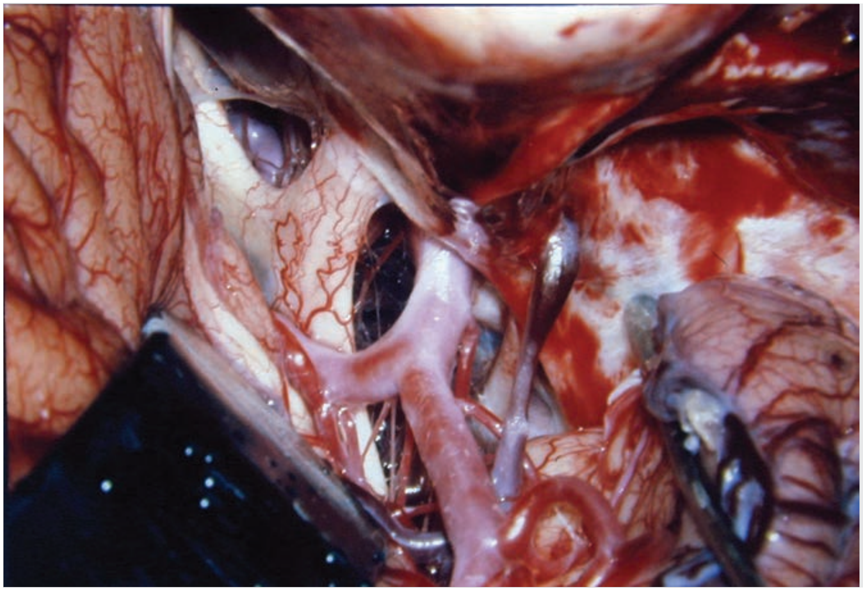

Fig. 13.5 The interpeduncular cistern can be accessed between the internal carotid artery and the optic nerve, the internal carotid artery and the third nerve, from above the carotid bifurcation, and by retracting or opening the tentorium cerebelli lateral to the third nerve.

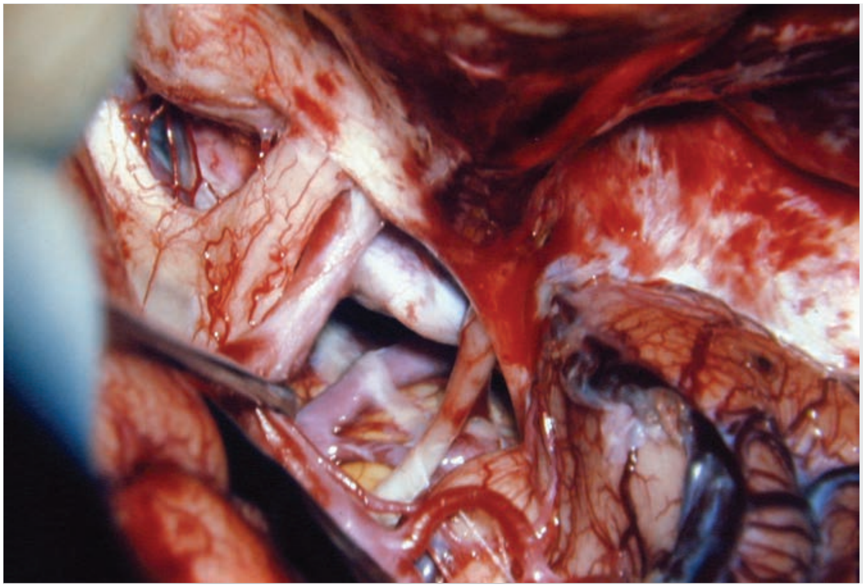

Fig. 13.6 The anterolateral route shows the interpeduncular cistern through the space between the carotid artery and the third nerve, exposing the basilar artery.

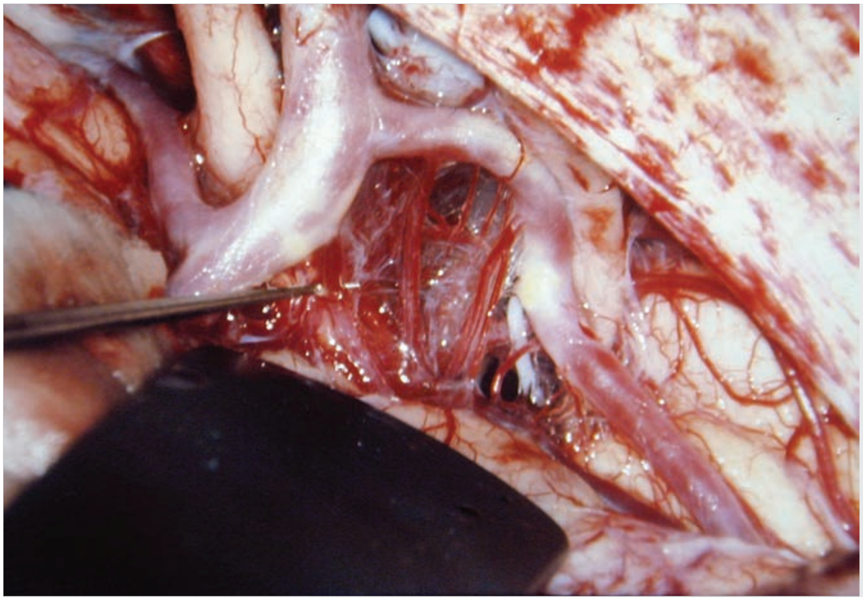

Fig. 13.7 Subtemporal route showing the posterior cerebral artery.

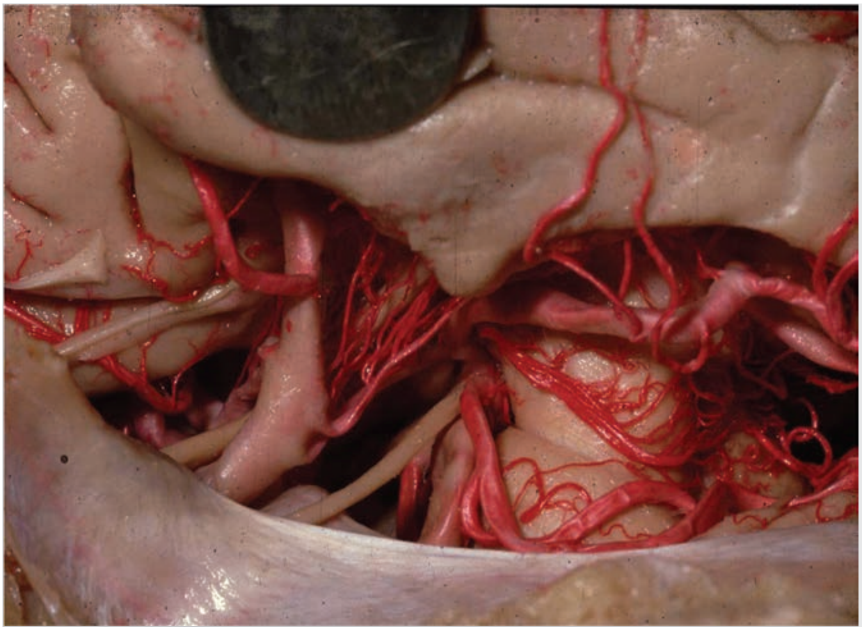

Fig. 13.8 Cadaveric specimen showing subtemporal view; the posterior communicating artery connects with the posterior cerebral artery. The superior cerebellar artery is below the third nerve and the posterior cerebral artery is above the third nerve.

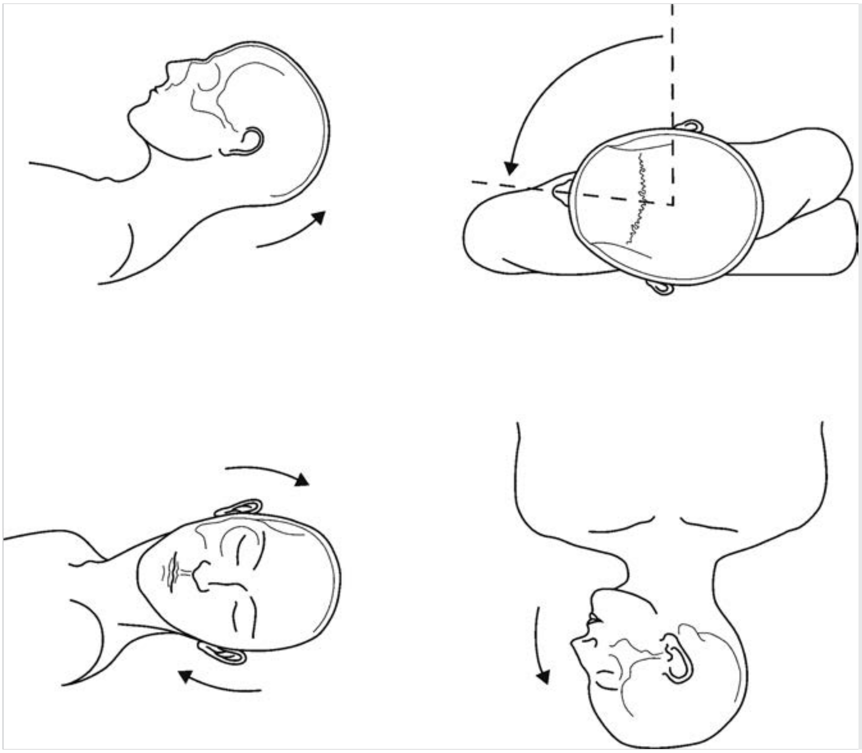

Fig. 13.9 The four important steps of patient positioning. After elevation, the head is carefully rotated 80 to 100 degrees to the contralateral side until the zygomatic arch is in a horizontal position. To facilitate this maneuver, the ipsilateral shoulder might be elevated by a cushion. Thereafter, the head is lateroflexed to the contralateral side to support the gravity-related self-retraction of the temporal lobe and allowing an ergonomic working position for the surgeon. The last step is to retroflex the head 15 to 20 degrees to prevent compression of the larynx and the ventilation tube.

which a **C**-shaped line is sawed from the burr hole to the anterior border of the first basal cut. The bone flap should be 15 to 25 mm wide and 15 to 20 mm high. After removal of the bone flap, the inner edge of the basal cut of the craniotomy is drilled flush with the floor of the middle fossa (▶ Fig. 13.15). Drilling or resection of a part of the zygomatic arch is usually not necessary. The operating microscope is brought in and the dura is opened in a **C**-shaped fashion with its base toward the middle fossa floor (▶ Fig. 13.16).

After the dura is opened, sufficient drainage of cerebrospinal fluid is the key to successful surgery because it allows gravity to allow the temporal lobe to fall away from the floor of the middle cranial fossa. The first step of the intradural procedure should be exposure and opening of the ambient cistern (▶ Fig. 13.16 and ▶ Fig. 13.17). If the intracranial pressure is high, such as in cases of SAH, the lateral ventricle should be punctured and a ventricular drain left in place. The retraction of the temporal lobe should be slowly increased while the line of retraction is first aimed slightly downward, then across the floor of the middle cranial fossa, and then upward to the tentorial edge. As the uncus is raised with the tip of the retractor, the opening into the interpeduncular cistern is exposed revealing

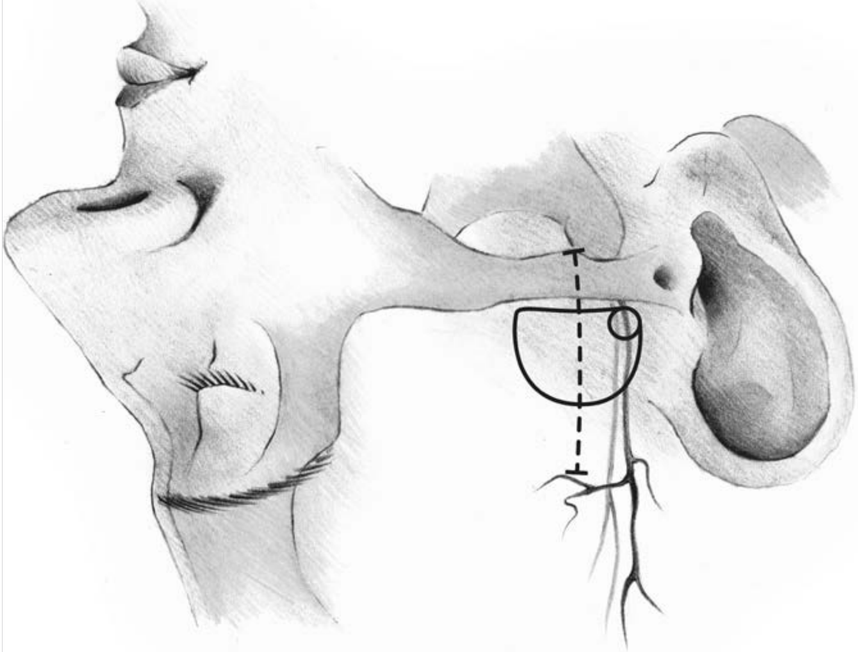

Fig. 13.10 The right temporal area from the surgeon's view. For preoperative orientation, the anatomical landmarks of the temporal skull as the lateral orbital rim, the zygomatic arch, the external auditory meatus, and the mastoid process are precisely defined. Note the course of the superficial neurovascular structures of the preauricular temporal region such as the superficial temporal artery and the auriculotemporal nerve in relationship with the skin incision and the size of the craniotomy.

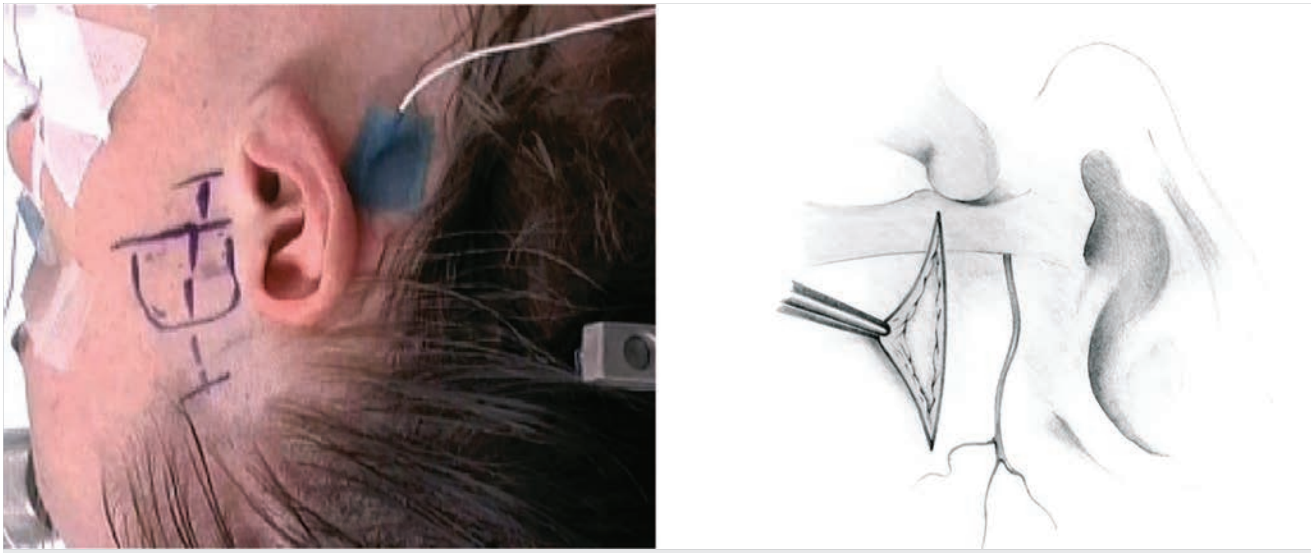

Fig. 13.11 The skin incision is performed at a right angle to the zygomatic arch ~1 cm in front of the external auditory meatus. The subcutaneous tissue is carefully dissected to preserve the superficial temporal artery and the auriculotemporal nerve as well as the upper temporal branch of the facial nerve.

the laterobasal aspect of the virtual suprasellar pyramid. The entrance of the interpeduncular fossa is narrow and divided by the third nerve into two windows. The opening can be widened substantially by retracting the tentorium. This is done by placing a 4–0 suture in front of the entrance and intratentorial course of the fourth nerve and then through the dura of the floor of the middle fossa. After dissection of the arachnoid and further removal of cerebrospinal fluid, the basilar bifurcation complex with its perforators, along with the ipsilateral and contralateral posterior communicating, P1, and ipsilateral more distal posterior cerebral arteries, can be visualized (▶ Fig. 13.17).

During clipping, temporary occlusion of the basilar artery may be necessary and can decrease tension in the aneurysm. Another method to do this is by endovascular balloon occlusion of the basilar artery during surgery using a catheter placed before surgery. Intraoperative angiography is also beneficial to demonstrate that the aneurysm is clipped and the major adjacent arteries remain patent.

13.5 Pretemporal Operative Technique

The patient is positioned supine with the head elevated above the heart to improve the venous return. The head is rotated contralaterally around 10 degrees and extended around 15 degrees

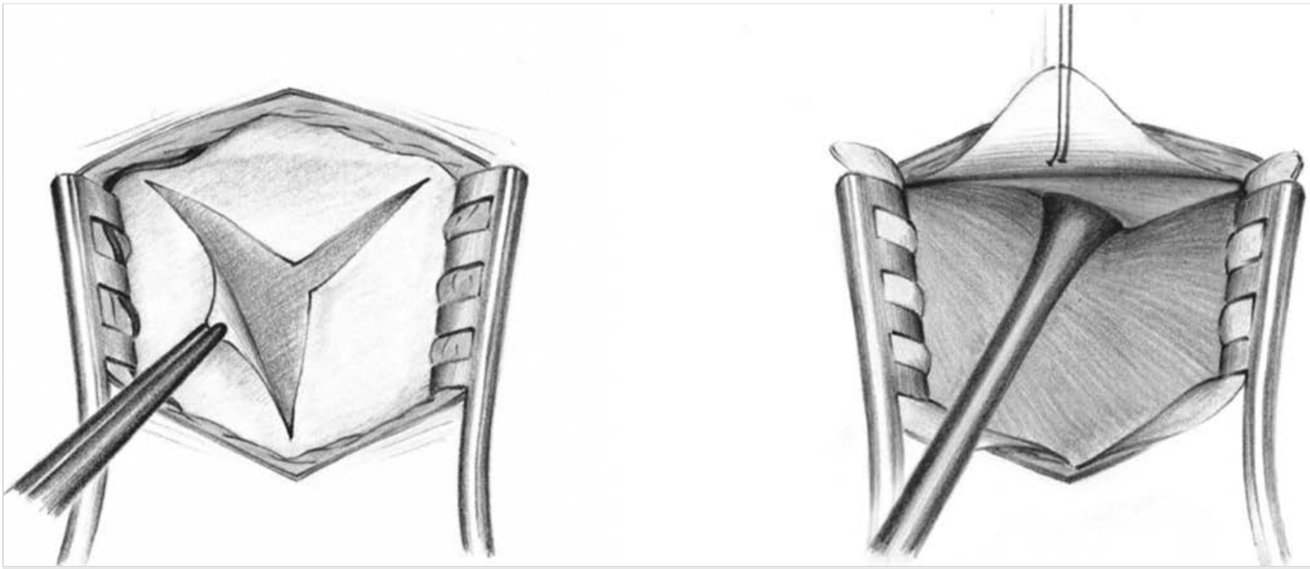

Fig. 13.12 After bilateral retraction of the skin, the fascia of the temporal muscle is incised in a **Y**-shaped fashion. The basal flap of the fascia is reflected downward over the zygomatic arch and fixed with a strong suture. The remaining two flaps are retracted bilaterally. Under the zygomatic arch, the inferior margin of the temporal muscle is mobilized bluntly and retracted upward.

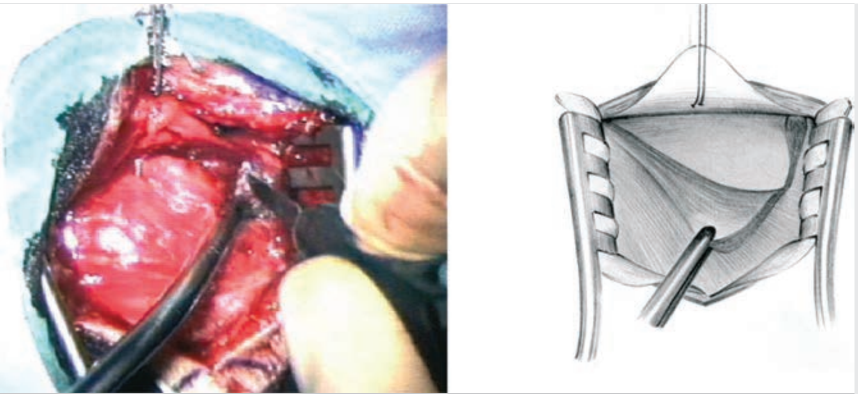

Fig. 13.13 In most of the cases with a sturdy temporal muscle, complete mobilization of the muscle is not possible without a small vertical incision at the posterior margin. Note the tiny groove between the protuberances of the medial and inferior temporal gyrus.

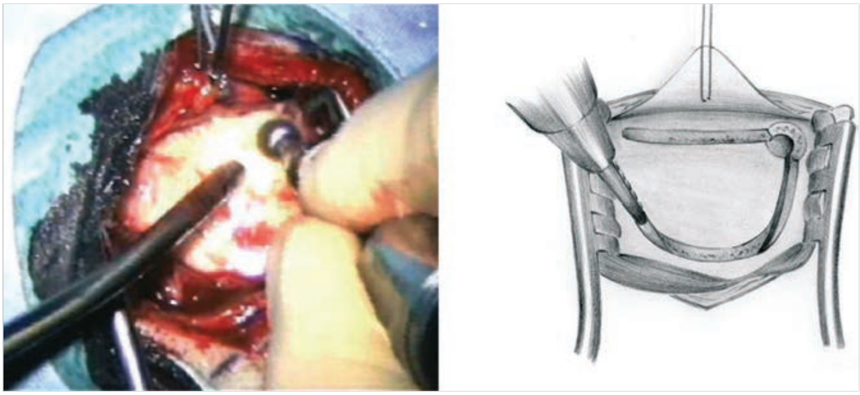

Fig. 13.14 The temporal muscle is retracted bilaterally and the squamous bone is exposed. After burr hole trephination, a straight line parallel to the zygomatic arch is sawed, followed by a **C**-shaped line from the burr hole to the anterior line of the previously performed temporobasal line. The size of the created bone flap by this means is ~15 to 25 mm wide and 15 to 20 mm high.

bringing the malar eminence to the highest point of the operative field. The skin incision starts at the anterior segment of the tragus, extends above the ear, and makes a curve toward the midline behind the hair. The interfascial dissection is done to preserve the frontal branch of the facial nerve. The temporalis muscle is detached from the entire zygomatic bone and reflected over the horizontal portion of the zygomatic arch.

A fronto-temporo-sphenoidal craniotomy is done with one, two, three, or four burr holes depending on the adherence between the dura and the bone (▶ Fig. 13.2). The first burr hole is made immediately below the most anterior limit of the superior temporal line and close to the zygomatic process of the frontal bone, the key hole. The second burr hole is located on the frontal bone, 2 cm medial to the first one and above the

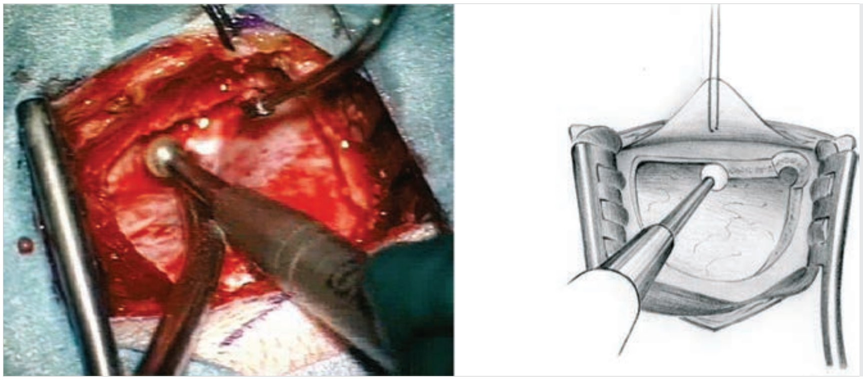

Fig. 13.15 An important step of the craniotomy after the bone flap is removed is the drilling of the inner edge of the temporobasal line using a high-speed drill under protection of the dura to expand the angle for visualization and manipulation in the depth.

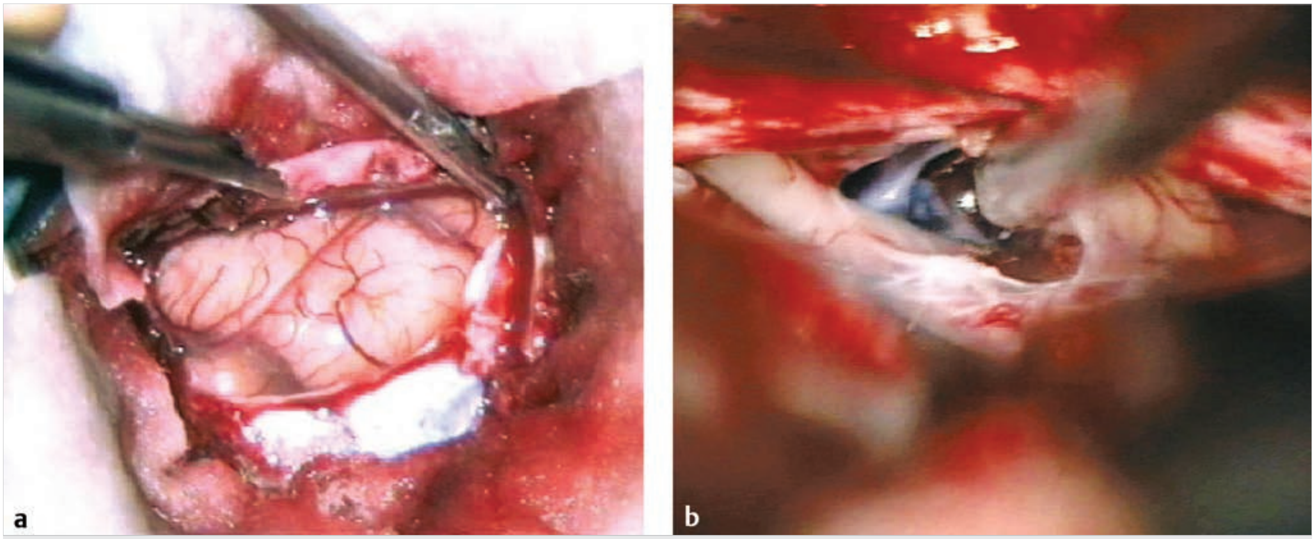

Fig. 13.16 **(a)** The dura is opened in a semicircular shape with its base toward the temporal base. The dural flap is fixed downward with two or three sutures. The temporal lobe is retracted carefully until the ambient cistern can be opened to release a certain amount of cerebrospinal fluid. **(b)** The entrance into the interpeduncular cistern between the tentorial edge and the oculomotor nerve is exposed.

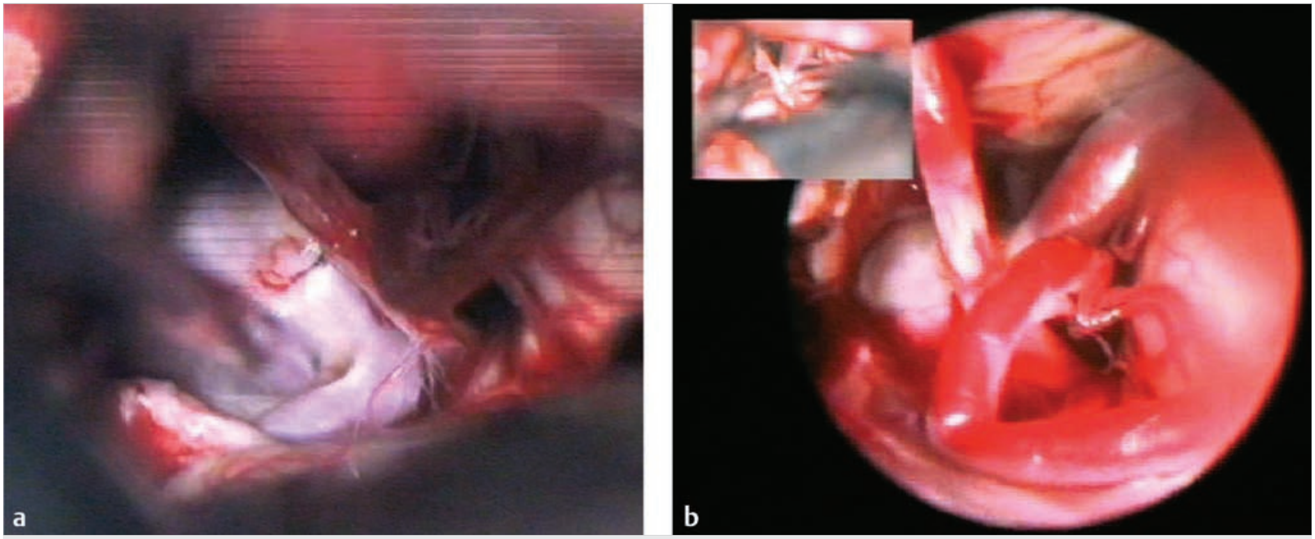

Fig. 13.17 **(a)** Microsurgical and **(b)** endoscopic views into the interpeduncular cistern. Note the course of the oculomotor nerve and the superior cerebellar artery and the posterior cerebral artery proceeding around it posteriorly. The aneurysm in this case projects anteriorly.

superior orbital rim. The third burr hole is below the superior temporal line at least 4.5 cm posterior to the first one. A fourth burr hole is created over the squamous part of the temporal bone at the level of the root of the zygomatic arch. The greater wing of the sphenoid and the squamosal part of the temporal bone are drilled out in order to expose the entire pole of the temporal lobe anteriorly and inferiorly. The orbital roof and the lesser sphenoid wing are drilled too. A great amount of bone resection provides a better visualization of the anterior and inferior portions of the temporal lobe and reduces the need for brain retraction (▶ Fig. 13.2 and ▶ Fig. 13.3).

The dura is opened in a curved incision from the frontal region to the level of the dural impression made by the sphenoid ridge. After that, the incision becomes straight and proceeds anteriorly toward the orbitomeningeal artery. In order to complete the exposure of the temporal lobe, another incision is made laterally and posteriorly following the contours of the craniotomy.

Microsurgical techniques are used to access the interpeduncular cistern (▶ Fig. 13.4 and ▶ Fig. 13.5). The bridging veins draining the temporal pole to the sphenoparietal sinus and the veins from the orbital surface of the frontal lobe to the sphenoparietal and cavernous sinuses are sacrificed. The arachnoid that binds the uncus to the oculomotor nerve and to the tentorial edge is opened. In order to mobilize the temporal lobe adequately, the ambient cistern and the arachnoid are dissected. After the cisternal opening, the temporal pole can be elevated superiorly and posteriorly to expose the interpeduncular region.

The deeper arachnoid membranes including the membrane of Liliequist are opened and dissection proceeds by sharply opening the arachnoid and exposing the basilar bifurcation and proximal posterior cerebral and superior cerebellar arteries, the aneurysm neck, and adjacent perforators (▶ Fig. 13.6, ▶ Fig. 13.7, ▶ Fig. 13.8). The principles of aneurysm microsurgery are followed. Verification of adequate aneurysm clipping and patency of adjacent arteries by indocyanine green videoangiography or catheter angiography is highly recommended.

13.6 Postoperative Management Including Possible Complications

This is similar to postoperative management for all aneurysm surgeries. Patients are transferred to the neurosurgical intensive care unit for at least 24 hours postoperatively. A computed tomography scan is obtained in the first few days. If intraoperative angiography was not done, we do a postoperative angiogram while the patient is in the hospital.

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14 Transsylvian Transclinoidal and Transcavernous Approach for Basilar Bifurcation Aneurysms

Ali F. Krisht

Abstract
Aneurysms arising from the basilar apex are usually treated endovascularly. Surgical approaches include subtemporal and pterional routes, the latter having many variations and extensions. Some expanded approaches widen the superficial part of the exposure whereas the transcavernous transclinoidal method opens up the depth of the surgical field and should increase the surgeon's ability to adequately clip basilar aneurysms. This approach is particularly useful for basilar aneurysms that are large or giant, project posteriorly, have a wide dysmorphic neck, or are associated with a low bifurcation or basilar dolichoectasia.

Keywords: Intracranial aneurysm, basilar apex, transcavernous, subarachnoid hemorrhage, clinoidectomy

14.1 Patient Selection

14.1.1 Indications for Surgery and Alternatives

Options for treatment of basilar apex region aneurysms include surgical clipping or endovascular treatment with coils. The preference for endovascular treatment tends to be even greater for basilar apex aneurysms because of the historically higher surgical morbidity with posterior circulation aneurysms compared with anterior circulation aneurysms, the establishment of Guglielmi detachable coils as an accepted modality for treating aneurysms in general, and the diminishing number of neurosurgeons with experience in the treatment of complex and posterior circulation aneurysms.

On the other hand, endovascular therapy has a high chance of failure in large aneurysms with wide necks. It also fails more commonly in aneurysms with a hemodynamic configuration, such as those at the basilar bifurcation region. The recanalization rate after endovascular therapy in general ranges between 20 and 30%, and it may be even higher with basilar apex aneurysms. In addition, the chance of future bleeding after coiling of basilar aneurysms is as high as 1 to 2% per year in some series.

The decision to treat basilar apex aneurysms endovascularly or with open surgery should take these features into consideration, as well as the aneurysm neck width, size, presence of intraluminal thrombus, location in relation to the posterior clinoids, and direction of projection of the aneurysm. The author has made extensive use of the transcavernous transclinoidal approach, which provides a much wider exposure at the depth of the surgical field and has helped achieve a more perfect clipping of the aneurysm, decreased the chance of recanalization, and improved the durability of treatment. This approach is indicated for basilar aneurysms that are large or giant and have a posteriorly projecting aneurysm dome (▶ Fig. 14.1), low bifurcation, wide dysmorphic base, and dolichoectasia (▶ Fig. 14.2). It is not indicated for small aneurysms that have small necks and project anteriorly above the level of the posterior clinoids (▶ Fig. 14.3).

14.1.2 Contraindications for Surgery

Patients with large basilar apex aneurysms who have a life expectancy of less than 5 years are considered only for endovascular therapy. Otherwise, patients are offered endovascular and microsurgical clipping and are informed of the risks and benefits of each to help them make their own decision.

14.1.3 Timing of Surgery

Although there are no strict rules and the decision is usually individualized, we use some guidelines based on the Yasargil subarachnoid hemorrhage (SAH) grading system (▶ Table 14.1). Patients who are grades 1 to 3 with associated hydrocephalus are operated on as soon as they present. Patients who are in grade 4 or worse are usually managed conservatively until their condition improves, after which treatment is initiated.

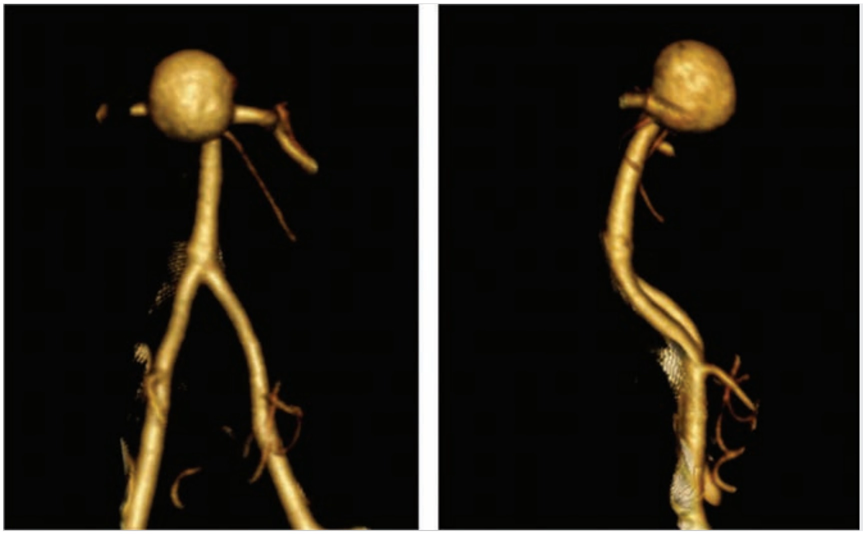

Fig. 14.1 Anteroposterior and lateral views of a large, wide-based, complex, posteriorly projecting basilar apex aneurysm.

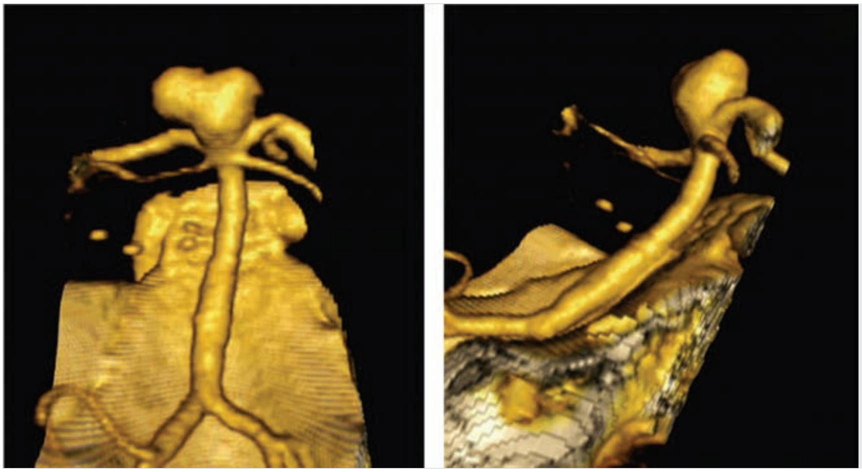

Fig. 14.2 Computed tomography angiography of a wide-based, dysmorphic, basilar apex aneurysm.

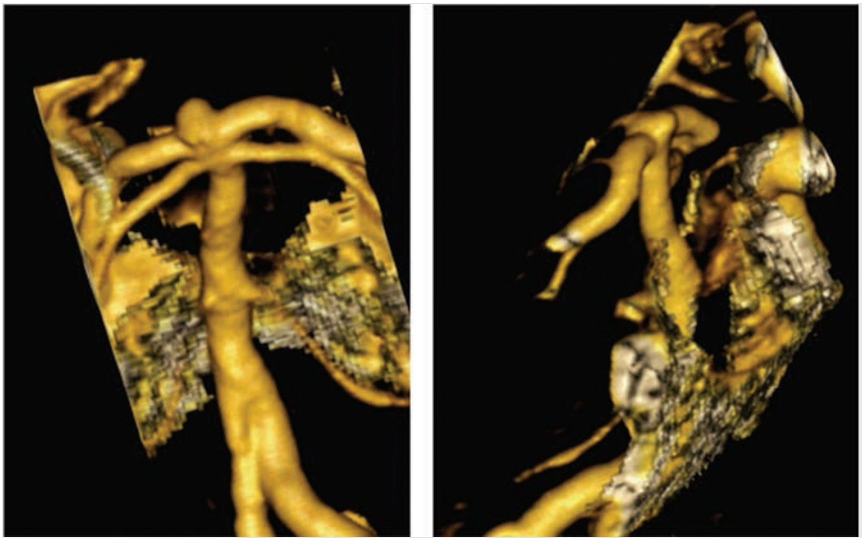

Fig. 14.3 Computed tomography angiography of a small basilar apex aneurysm located above the posterior clinoid process not needing the transcavernous approach.

Table 14.1 Yasargil subarachnoid hemorrhage grading system

Grade	Condition	Deficit, cranial neuropathy, or A or B hemisyndrome	
0 (unruptured)	Asymptomatic	A	No
	Symptomatic	B	Yes
1 (SAH)	No deficits	A	No
	Minimal deficits such as cranial neuropathy	B	Yes
2 (SAH)	Headache and meningismus	A	No
	Minimal deficits	B	Yes
3 (SAH)	Lethargic, confused, combative	A	No
	With hemisyndrome	B	Yes
4 (SAH)	Semicomatose, respond to pain but not voice		
5 (SAH)	Comatose, no reaction, failing vital signs		

Abbreviation: SAH, subarachnoid hemorrhage.

14.2 Preoperative Preparation

An arterial line is inserted in patients with SAH upon admission to the intensive care unit. Blood pressure is controlled in awake patients with a β-blocker such as atenolol (25 mg orally) or a clonidine patch (0.1–0.2 mg) or both. This will usually adequately control the blood pressure without the need for intravenous agents. In patients who have significant ventriculomegaly, an external ventricular drain may be inserted and kept at 15 cm above the level of the external auditory meatus. This is usually done in patients who are grade 3 or 4 with evidence of ventriculomegaly because it can results in significant clinical improvement, leading the author to recommend immediate surgery. Perioperative antibiotics, hydrocortisone, and an anticonvulsant such as phenytoin are administered and continued postoperatively.

In addition to catheter angiography, a computed tomography angiography (CTA) is obtained in most patients as a baseline with which to compare postoperative CTA. We often obtain a magnetic resonance imaging scan with perfusion and diffusion images in patients who are grade 3 or worse. This detects ischemic changes before surgery and may be an indication to delay surgery. One etiology is SAH within the week or so before the current presentation, leading to vasospasm and ischemia.

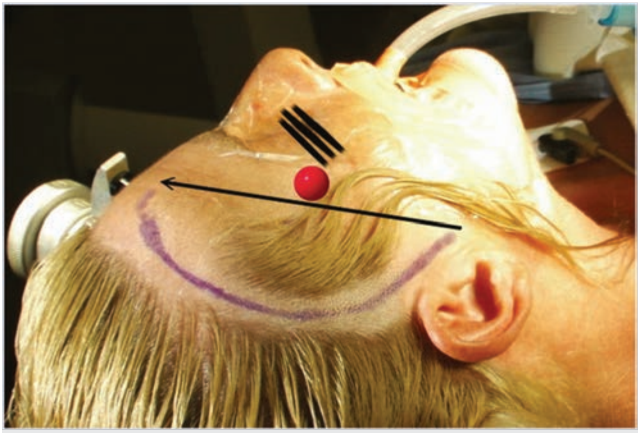

Fig. 14.4 The head position and the marking of the skin incision. The *arrow* connects the beginning and the end of the incision, which illustrates how the base of the flap will reach the junction of the frontal zygoma to the orbital rim (*red ball*).

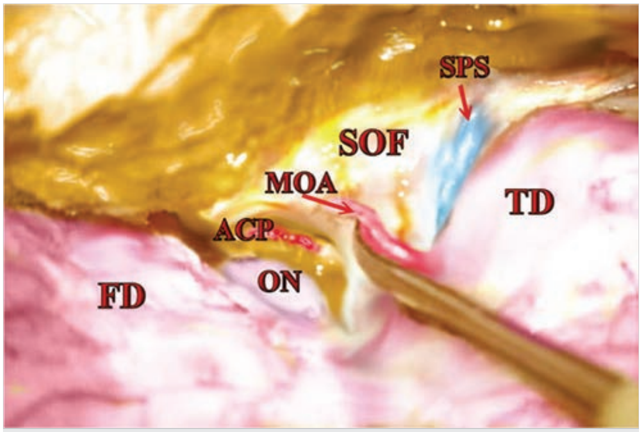

Fig. 14.5 The exposure of the meningo-orbital artery (MOA), connecting the temporal dura to the periorbita of the superior orbital fissure (SOF). ACP, anterior clinoid process; FD, frontal dura; ON, optic nerve; SPS, sphenoparietal sinus; TD, temporal dura. Note the temporal extension of the craniotomy exposing the dura at the temporal tip.

We monitor somatosensory evoked potential and brainstem evoked responses during surgery. Changes in evoked potentials are an indication to remove temporary clips, if placed, and the clipping process should be adjusted accordingly.

14.3 Operative Procedure

14.3.1 Skin Incision and Craniotomy

Under general endotracheal anesthesia, the patient is placed in a supine position and the head is turned, usually to the left side. Preparation is made for a frontotemporal craniotomy with pretemporal extension. The hair is shaved along a strip where the incision will be made (▶ Fig. 14.4). After scrubbing and draping the wound in a sterile fashion, the skin incision is made, and hemostasis is established with scalp clips.

The flap is usually designed to extend from the preauricular region just anterior to the tragus, in a curvilinear fashion, to the level of the perimedian midsagittal plane. A straight line extending from the beginning of the incision to its end should cross over the junction of the frontal zygoma with the orbital rim (▶ Fig. 14.4). This ensures that when the flap is reflected inferiorly enough pretemporal exposure can be achieved. The scalp flap is reflected using sharp dissection with a knife. A small pericranial flap is elevated and prepared in case the frontal sinus is opened. The deep fascia of the temporalis muscle is dissected away from the zygomatic arch. The zygomatic arch is trimmed and the temporalis muscle is cut away from both the frontal and temporal zygomatic roots. A cuff of fascia is left along the superior temporal line to resuture the muscle back during closure.

A frontotemporal bone flap similar to that for a pterional approach is raised, except with a more temporal extension. After the flap is raised, the microscope is introduced, and under magnification, the sphenoid wing, including the region at the junction of the orbital rim with the zygoma, is drilled flat with the orbital roof. The most posterior third of the roof and the lateral wall of the orbit are drilled to an eggshell thickness and removed with a small rongeur. The bone is removed on both sides of the meningo-orbital artery connecting the pretemporal dura with the periorbital tissue (▶ Fig. 14.5). On the medial side of the meningo-orbital artery, the bone is removed with rongeurs to the level of the lateral aspect of the anterior clinoid process. On the lateral aspect of the meningo-orbital artery, the bone is removed all the way to the level of the superior orbital fissure. The meningo-orbital artery is coagulated and cut. Its location is a point used to start dissection of the dura propria over the temporal lobe from the lateral wall of the cavernous sinus (▶ Fig. 14.6). The dissection is continued in this plane exposing the third, fourth, and fifth cranial nerves within the lateral wall of the cavernous sinus (▶ Fig. 14.7). Because of the possible need to open the cavernous sinus posteriorly at the petroclival junction and over the posterior clinoid process, fibrin

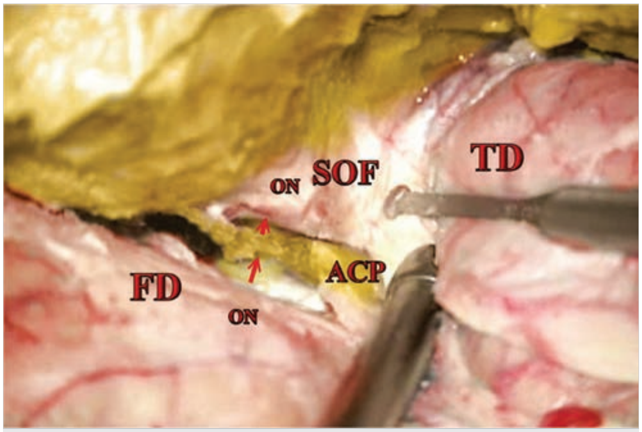

Fig. 14.6 After clipping the meningo-orbital artery, the dura propria of the temporal lobe is being separated from the lateral wall of the cavernous sinus starting from the level of the superior orbital fissure (SOF). This figure also shows the most medial extension of the sphenoid wing at the level of the anterior clinoid process (ACP), as well as both the extradural and the intraorbital course of the optic nerve (ON). FD, frontal dura; TD, temporal dura.

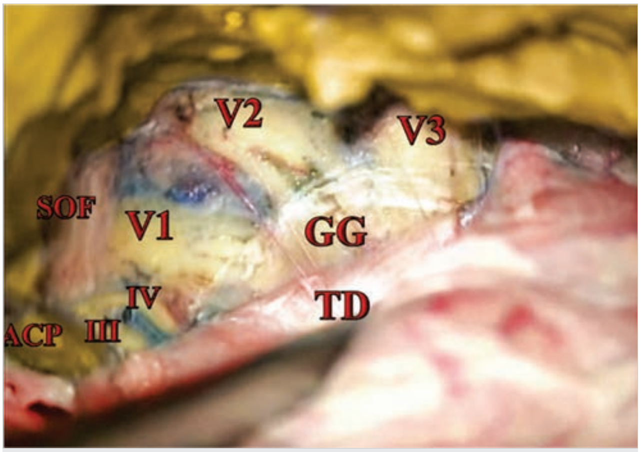

Fig. 14.7 Pretemporal exposure of the lateral wall of the cavernous sinus. ACP, anterior clinoid process; GG, gasserian ganglion; III, ocular motor nerve; IV, trochlear nerve; SOF, superior orbital fissure; TD, temporal dura; V1, V2, V3, branches of the trigeminal nerve.

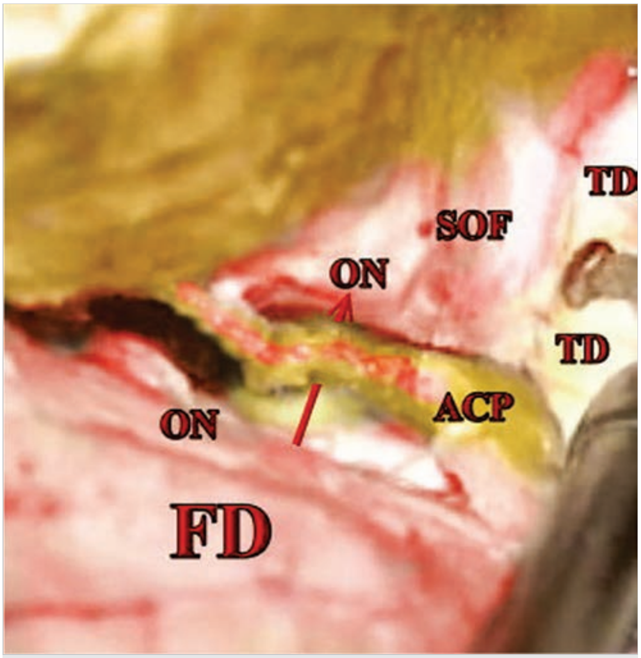

Fig. 14.9 The course of the optic nerve (ON) within the optic canal seen from both the extradural portion of the proximal part of the optic canal and the intraorbital portion within the orbit itself. Note how the anterior clinoid process (ACP) is better exposed due to reflection of the temporal dura (TD) away from the superior orbital fissure (SOF). This made the clinoid process more superficial in location and easier to drill. FD, frontal dura.

glue is injected into the cavernous sinus between the first and second divisions of the trigeminal nerve (▶ Fig. 14.8). This slows epidural oozing and helps further the dissection process. By peeling the dura away from the lateral wall of the cavernous sinus, the lateral aspect of the anterior clinoid process becomes more exposed and superficial. The optic nerve is visualized from the extradural entry point to the intraorbital exit point, which helps direct drilling and removal of the anterior clinoid process (▶ Fig. 14.9).

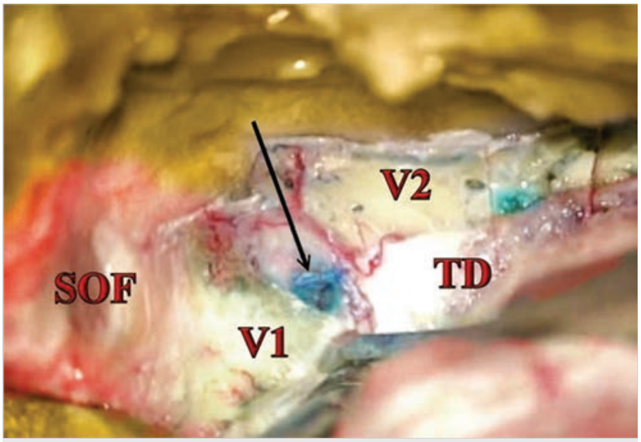

Fig. 14.8 The pretemporal exposure and the site of the injection of fibrin glue to establish hemostasis within the cavernous sinus between the V1 and V2 branches of the trigeminal nerve. SOF, superior orbital fissure, TD, temporal dura.

14.3.2 Removal of the Anterior Clinoid Process

The anterior clinoid process has three connections. One is to the orbital roof, which should already have been removed. The second is to the roof of the optic canal, and the third is to the floor of the optic canal (the optic strut). At this stage, the anterior clinoid is removed using a high-speed diamond drill under copious irrigation. A thin shell of remaining bone can be removed with a rongeur. The optic strut is drilled, avoiding the clinoidal segment of the internal carotid artery.

14.3.3 Dural Opening

The dura is opened in a **T**-shaped fashion with the vertical arm of the T along the indentation of the sphenoid wing (▶ Fig. 14.10). The dural opening is directed toward the oculomotor trigone region whereby the dural fibrous bands over the oculomotor canal are cut. This allows exposure of both the intra- and extradural portion of the oculomotor nerve. Further dissection of the oculomotor nerve from the fibrous attachments along both its lateral and medial gutters is done to maximize its mobilization. In patients with prominent temporal polar veins, the dural incision is extended at a more inferior level along a lateral and more temporal incision to help preserve the dura over the temporal lobe when the exposure is made to the interpeduncular fossa. Following this, a triangular dural leaflet is removed extending laterally from the oculomotor trigone to the level of the optic nerve dura medially and along the internal carotid dura ring. This allows direct exposure of the posterior clinoid process in case it has to be removed, as for low-lying basilar bifurcations.

14.3.4 Intradural Dissection

At this stage, the arachnoid over the optic and chiasmatic cisterns is opened. Cerebrospinal fluid is suctioned, which relaxes the brain. The sylvian fissure is opened widely from inside to outside. A self-retaining brain spatula is placed on the temporal

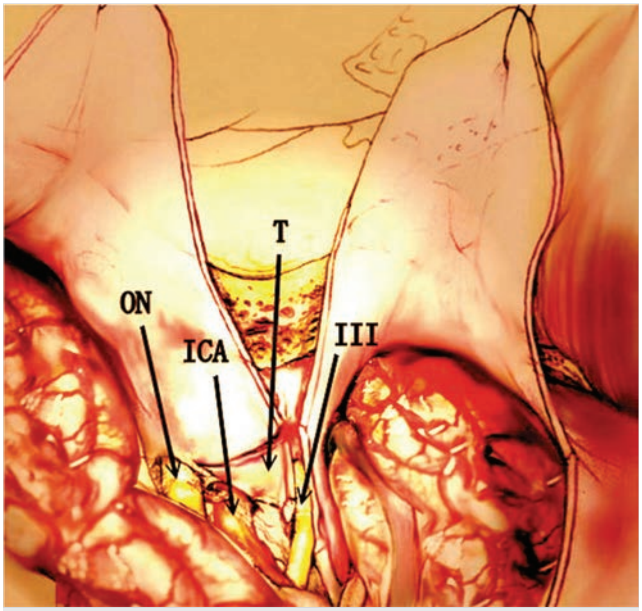

Fig. 14.10 The dural incision along the indentation of the sphenoid wing. The triangular flap (T) extending from the optic nerve (ON) to the ocular motor nerve (III) is seen. This flap is removed to allow exposure of the posterior clinoid process and the region of the interpeduncular fossa. Note the dura opening along the oculomotor trigone exposing the third nerve (III) from its intradural portion to its extradural portion, along the lateral wall of the cavernous sinus. ICA, internal carotid artery.

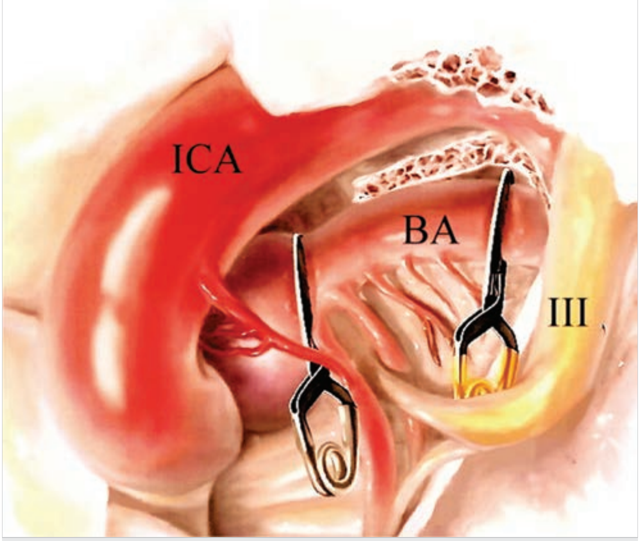

Fig. 14.12 The expected exposure after removal of both the anterior and the posterior clinoid processes. The basilar artery trunk is exposed (BA) and the temporary clip is applied to a perforator-free zone. This allows collateral blood flow from the superior cerebellar artery. Note the distance between the temporary clip and the permanent clip, which allows better maneuverability and exposure of the aneurysm at its neck. III, oculomotor nerve; ICA, internal carotid artery.

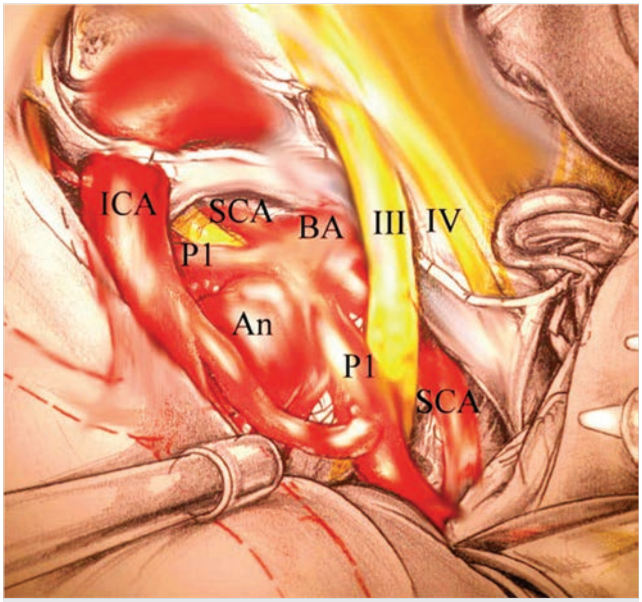

Fig. 14.11 The intraoperative wide exposure of the basilar apex region. An, aneurysm; BA, basilar trunk; ICA, internal carotid artery; III, oculomotor nerve; IV, trochlear nerve; P1, posterior communicating artery; SCA, superior cerebellar artery.

lobe, which is retracted posteriorly and laterally. The membrane of Liliequist is opened and the arachnoid above and below the third nerve is dissected. The postcommunicating segment of the posterior cerebral artery is visualized and followed medially to the basilar bifurcation. If the bifurcation is low, the posterior clinoid process may need to be removed. This is done by cutting the petroclival dural fold and exposing the posterior medial extension of the cavernous sinus. The prior injection of the sinus with fibrin glue should prevent any bleeding. The dura is further peeled off the posterior clinoid process. A bone ultrasonic aspirator is used to shave the lateral aspect of the posterior clinoid process from underneath the third nerve. Then, the third nerve is mobilized to a more lateral position and the medial extension of the posterior clinoid process is removed. This step exposes the basilar trunk below the takeoff of the superior cerebellar arteries and at a perforator-free zone. It will also help expose the contralateral precommunicating (P1 segment) segment of the posterior cerebral artery (▶ Fig. 14.11).

14.3.5 The Clipping Process

Clipping is generally done with a temporary clip applied to the basilar trunk (▶ Fig. 14.12). We do not use additional cerebral protection agents during temporary clipping but maintain normovolemia and normal blood pressure. The optimal location for the temporary clip is proximal to the superior cerebellar arteries at a perforator-free zone. We limit temporary clipping to 2-minute epochs if possible.

The location and size of the ipsilateral posterior communicating artery are assessed next. If it is small and obstructs the view of the aneurysm neck, it can be coagulated and cut at a perforator-free zone. The neck of the aneurysm is dissected and the thalamoperforating arteries are identified so they can be preserved. When one is ready to clip the aneurysm, a temporary clip is applied to the basilar trunk and the initial permanent clip is applied. A straight clip is used for aneurysms projecting anteriorly or superiorly. For posteriorly projecting aneurysms, the posterior aspect of the neck may be obstructed by the ipsilateral precommunicating segment (P1 segment) of the posterior cerebral artery. A fenestrated clip may be used in such cases to help lift the posterior wall of the aneurysm. This is followed by a straight clip applied anterior to the precommunicating segment of the posterior cerebral artery and distal to the first clip. The fenestrated clip is removed and the temporary clip is then removed. A Doppler ultrasound probe is used to check for flow in the aneurysm. If there is no flow and the aneurysm seems collapsed, dissection of both the anterior and posterior aspects of the aneurysm is made to verify that no perforators have been included in the clip blades. We use intraoperative angiography if there is any question about the adequacy of clipping or patency of adjacent arteries.

For large aneurysms, especially those projecting anteriorly or superiorly, with no thalamoperforating arteries adherent to the dome, bipolar coagulation on a 25-power setting is helpful to shrink the aneurysm dome. Posteriorly projecting aneurysms are more challenging because of frequent adherence of perforating arteries to the posterior aspect of the aneurysm. We have found there is always a window between the aneurysm neck and the takeoff of the perforators through which the aneurysm clip blades can be introduced.

14.3.6 Wound Closure

The dura is approximated using 4–0 Neurolon (Ethicon, Inc., Somerville, NJ) sutures in a watertight fashion. The defect over the oculomotor trigone and anterior clinoid space is plugged with pieces of temporalis muscle. The closure is further reinforced with DuraGen (Integra LifeSciences, Plainsboro, NJ), TISSEEL (Baxter, Deerfield, IL), or fibrin glue. Following this, the pretemporal space is obliterated by applying tack-up stitches to it as well as to the dural edges. The bone flap is replaced with a cranial fix device or plates. The defect at the junction of the zygoma with the orbital rim is reconstructed with a bone substitute. Scalp flap closure is routine.

14.4 Postoperative Management Including Possible Complications

Patients with unruptured aneurysms are kept in the intensive care unit overnight and then transferred to a regular floor, put on a regular diet, and allowed to increase their activities to full ambulation within 24 hours. They are usually discharged 3 to 4 days after surgery. Patients with SAH are kept in the intensive care unit and monitored for the occurrence of vasospasm. We use CTA in many cases instead of postoperative catheter angiography as well as to monitor for vasospasm.

15 Vertebral Artery and Posterior Inferior Cerebellar Artery Aneurysms

Amr Abdulazim, Daniel Hänggi, and Nima Etminan

Abstract
Surgical management of common saccular vertebral and posterior inferior cerebellar artery (PICA) aneurysms usually requires a lateral or midline suboccipital approach. As for nonsaccular/fusiform PICA aneurysms, reconstructive surgical measures should be taken into consideration.

Keywords: intracranial aneurysm, posterior inferior cerebellar artery, vertebral artery

15.1 Introduction

Compared with anterior circulation aneurysms, aneurysms arising from the vertebral artery (VA) and/or posterior inferior cerebellar artery (PICA) are rather rare and account for less than 2% of all aneurysms.[1,2,3] Repair of VA/PICA aneurysms is commonly performed by endovascular strategies but surgical repair may be required in selected scenarios. The common saccular aneurysms arising between the origin of the PICA and the proximal VA (proximal PICA aneurysms, segments I–III) usually require only a lateral suboccipital approach with removal of the rim of the foramen magnum. Distal (segments IV and V) PICA aneurysms are also best approached by the midline suboccipital approach. In case of a distal PICA origin from the VA, a lateral and superior extension could be necessary but the previously proposed lateral transcondylar suboccipital approach is rarely required. In cases of nonsaccular/fusiform PICA aneurysms requiring reconstructive measures (e.g., end-to-end anastomosis after resection of the aneurysm, PICA–PICA side-to-side anastomosis, or occipital artery-to-PICA bypass), options should be discussed within the neurovascular team before destructive measures, such as parent vessel occlusion (especially of the dominant PICA), are considered.

15.2 Anatomical Features and Patient Selection

The posterior inferior cerebellar artery (PICA) has a highly variable origin: in 90% of individuals, it originates from the so-called intradural V4 segment of the vertebral artery (VA), and in about 10% it originates extracranially from the extradural V3 segment of the VA, or from the basilar artery. Anatomically, the PICA runs in a tortuous fashion around the lateral medulla oblongata at the level of the caudal cranial nerves (IX–XII). At the anterior aspect of the cerebellar tonsil, it forms the caudal loop. In its further course, the PICA runs between the dorsal aspect of the medulla oblongata and the tonsils, and forms the cranial loop. The distal portion divides into two main branches to supply the vermis and cerebellar hemisphere.[4] Thus, based on its anatomical course in relation to the posterior fossa structure, the PICA can be divided into five segments:

- Anteromedullary (segment I).
- Lateromedullary (segment II).
- Tonsillomedullary (segment III).
- Telovelotonsillary (segment IV).
- The cortical segment (segment V).

The rami perforantes or perforating arteries arise from the first three segments to supply the posterolateral medulla. The first three segments are also referred to as the proximal PICA; segments IV and V are the distal PICA. About 2% of all intracranial aneurysms are associated with the PICA. Clinically, PICA aneurysms may present with rupture, which is virtually always subarachnoid hemorrhage (SAH). Intraventricular hemorrhage is common as well. These aneurysms also may present with signs of ischemia or with mass effect with symptoms such as hiccups, dysphagia, and other paralytic caudal cranial nerve palsies.

In general, initial diagnostic workup should include standard blood laboratory tests and cranial computed tomography (CCT) for confirmation and localization of the hemorrhage and also CT angiography, which helps to distinguish proximal from distal PICA origins, in relation to the cranial base.

Catheter rotational digital subtraction angiography with three-dimensional reconstructions, however, remains the diagnostic method of choice to confirm presence of aneurysms, especially with respect to precise site, size, shape, and projection of the aneurysm, in relation to the perforating arteries. More importantly, catheter angiography gives information about the size of both PICAs and, indirectly, the size of their supply territory, which may be an important factor in decision-making regarding reconstructive versus destructive aneurysm repair. Following angiography, interdisciplinary consultation between the neurosurgeons and/or neuroradiologists should define the optimal mode of repairing the aneurysm, preferably by preserving the parent vessel. The decision will usually include aneurysm-related factors (morphology, size, projection, and location of the aneurysm in relation to the PICA aforementioned segments and of the PICA origin on the VA), patient-related factors (e.g., patient age, clinical status on admission, comorbid disease), and whether surgical evacuation of a hematoma or decompression may be required. Lastly, logistical aspects such as availability of endovascular and neurosurgical competence may play a role during decision-making.

Most PICA-origin aneurysms can be treated by either endovascular or microsurgical route, and there are no robust data to guide decision-making, since in the largest randomized clinical trial comparing clipping and coiling, only 2.6% of all aneurysms included were located in the posterior circulation and only 1.4% of all aneurysms included were PICA aneurysms (a total of 31 cases).[5] Additionally, small, single-center and retrospective studies report good outcomes with both treatment modalities, although these studies are not randomized and these types of reports typically underestimate complications (▶ Table 15.1). Many PICA-origin aneurysms are relatively broad based and therefore particularly complicated for endovascular therapy.[3] However, the main challenge of microsurgical treatment is the anatomical relation to the caudal cranial nerves and thus the risk of caudal cranial nerve deficits, especially swallowing and airway control.

Table 15.1 Overview of larger series (more than five patients) reporting on surgical and/or endovascular treatment of PICA aneurysms

Authors (year)	No. of patients (aneurysms)	Treatment modality	Aneurysm locations[a]	Aneurysm rupture status	Outcome[b]
Rodríguez-Hernández et al (2011)[4]	50 (51)	ST	35 proximal, 16 distal	100% ruptured	Good: 41; poor: 10
Tokimura et al (2011)[2]	22 (23)	ST (17), EVT (5)	13 proximal, 10 distal	100% ruptured	ST: good: 16; poor: 2 EVT: good: 4; poor: 1
Nourbakhsh et al (2010)[6]	15	ST (13), EVT (2)	10 proximal, 3 distal, 2 n.s.	100% ruptured	Good: 9; poor: 5; n.s.: 1
Peluso et al (2008)[7]	30	EVT	n.s.	100% ruptured	Good: 21; poor: 9
Al-khayat et al (2005)[8]	52	ST	52 proximal	64% ruptured, 36% unruptured	Good: 47; poor: 5
Horowitz et al (1998)[9]	38	ST	32 proximal, 6 distal	73% ruptured, 27% unruptured	Good: 33; poor: 2; n.s.: 2
Orakcioglu et al (2005)[10]	14 (16)	ST (13), EVT (1)	11 proximal, 5 distal	100% ruptured	Good: 12, poor: 2
Sandalcioglu et al (2005)[11]	16	ST (7), EVT (9)	13 proximal, 3 distal	100% ruptured	ST: good: 6; poor: 1 EVT: good: 6; poor: 3
D'Ambrosio et al (2004)[12]	17	ST	All proximal	100% ruptured	Good: 13; poor: 4
Kleinpeter (2004)[13]	11	ST	8 proximal, 2 distal	100% ruptured	Good: 7; poor: 3
Horiuchi et al (2003)[14]	18 (21)	ST	10 proximal, 11 distal	100% ruptured	Good: 16; poor: 2
Mukonoweshuro et al (2003)[15]	23 (24)	EVT	19 proximal, 5 distal	100% ruptured	Good: 19; poor: 3; n.s.: 1
Matsushima et al (2001)[16]	8	ST	n.s.	70% ruptured, 30% unruptured	Good: 4; CN palsy: 3; poor: 1
Bertalanffy et al (1998)[17]	5	ST	10 proximal, 2 distal	100% ruptured	Good: 8; poor: 4
Ishikawa et al (1990)[1]	5	ST	2 proximal, 3 distal	100% ruptured	Good: 2; poor: 3
Yamaura (1988)[18]	56	ST (43), conservative (13)	46 proximal, 9 distal, 1 n.s.	75% ruptured, 25% unruptured	ST: good: 43 Conservative: good: 3; poor: 10

Abbreviations: CN, cranial nerve; EVT, endovascular treatment; n.s., not specified; ST, surgical treatment.

[a] Aneurysm location corresponds to the segments of the PICA (proximal = I–III, distal = IV, V).

[b] Outcome was dichotomized according to the Glasgow Outcome Scale (GOS) and/or modified Rankin Scale (mRS) into poor (GOS 1–3, mRS 4–6) versus good (GOS 4–5, mRS 0–3).

For PICA-origin aneurysms, the most important factors in the initial interdisciplinary decision-making regarding treatment modality are: (1) the level of PICA origin in relation to the VA and the brainstem and (2) the shape of the aneurysm (i.e., broad- vs. narrow-necked). "High-lying" or prepontine aneurysm locations are difficult to approach microsurgically, and in these situations, the endovascular approach appears more favorable. On the other hand, a broad-based aneurysm—particularly if the PICA arises from the aneurysm—may be challenging for endovascular therapy and may be better repaired by microsurgical means. Access to proximal PICA aneurysms, i.e., segments I–III, is gained via a lateralized suboccipital craniectomy, with removal of the foramen magnum (▶ Fig. 15.1). Distal PICA aneurysms, i.e., segments IV–V, of the vermis or of hemispheric branches can be treated surgically with low morbidity, owing to their distal location that requires minimal or no manipulation of the cerebellum. Access is gained via a median suboccipital craniectomy with or without a laminectomy of the atlas (▶ Fig. 15.1).

15.3 General Preoperative Management

In patients suffering from SAH, avoidance of re-rupture is a primary objective following hospital admission. Patients with World Federation of Neurosurgical Societies (WFNS) grades II or higher should be monitored in a neurologic intensive care or similar unit. WFNS grade I patients can be admitted to a single room on the normal floor. In such patients, continuous recording of vital signs, including blood pressure and neurologic status, and avoidance of blood pressure fluctuations are important. Analgesia for headache, mild sedation, and stool softeners generally are prescribed prophylactically but painful procedures such as intravenous injections and insertion of arterial and central lines should be done under adequate analgesia or avoided. Patients who have been intubated for transport or medical procedures after admission should not

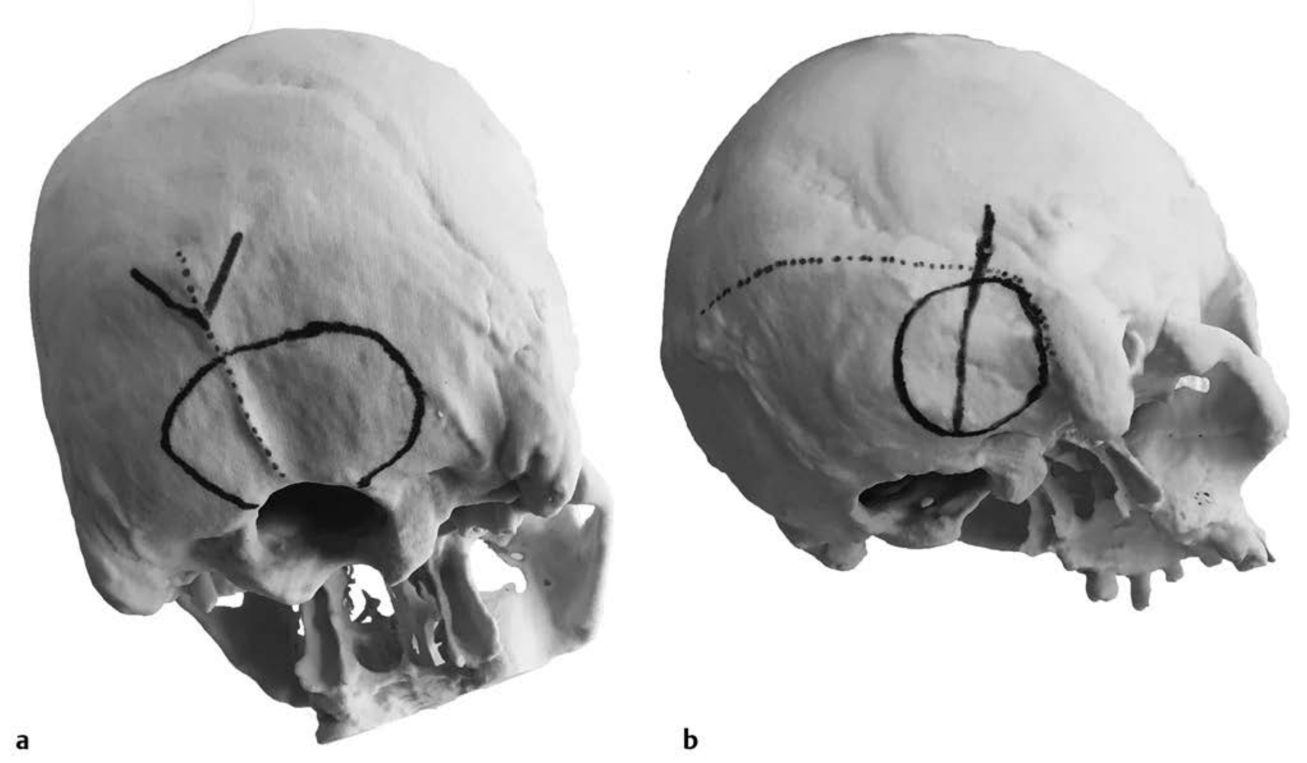

Fig. 15.1 Outline of the bony opening for a midline suboccipital craniectomy or craniotomy (**a**) to access, in this case, a right-sided distal PICA aneurysm and the bony opening for a lateral suboccipital craniotomy or craniectomy for aneurysms arising at the origin of the PICA from the VA (**b**).

be extubated until the ruptured aneurysm has been secured. Oral nimodipine, 60 mg every 4 hours, is recommended for good-grade patients and, where available, intravenous administration is necessary for unconscious patients. The dose needs to be reduced for patients weighing less than 60 kg or if arterial hypotension ensues (< 110 mg systolic).

Stronger analgesics (opioids) or sedatives should only be given under monitored conditions, particularly when administered intravenously.

For surgical repair of aneurysms in WFNS I or II patients without space-occupying hemorrhage or obstructive hydrocephalus, a lumbar cerebrospinal fluid (CSF) drain may be inserted after induction of anesthesia for brain relaxation, unless ventriculostomy via an external ventricular drain has been performed.

A sufficient level of anesthesia must have been obtained prior to fixation of the Mayfield clamp. We recommend mannitol at a dose of 1.0 g/kg bodyweight for either ruptured or unruptured aneurysm surgery to achieve additional brain relaxation and to minimize retraction, except in elderly patients, in whom the dosage should be reduced. Following craniotomy and opening of the dura, 50 to 100 mL of CSF should be drained from the lumbar or ventricular drain.

15.4 Operative Procedure

Because of the anatomical relation of PICA aneurysms to the caudal cranial nerves, electrophysiological monitoring (i.e., somatosensory and motor evoked potentials and electromyography of cranial nerves IX–XII) should be performed, where available.

15.4.1 Lateral Suboccipital Approach

Although a semisitting or sitting position may have its virtues, the park bench position is increasingly favored due to higher comfort for the surgeon. The shoulders should be rotated about 45 degrees away from the surgeon, to not limit access to the posterior fossa, and the head should be inclined as much as possible, to reduce the slope of the surgical trajectory.

The pins should be placed in a plane perpendicular to the incision. A linear, vertical 6- to 8-cm incision line is planned about 3 cm behind the mastoid fossa. The upper end of the incision corresponds to the upper edge of the ear helix, and the lower end corresponds approximately to near the spinous process of the axis. The occipital artery should be coagulated and divided. Obviously, it is necessary to spare and dissect the artery during skin incision if an occipital artery-to-PICA bypass is a planned option for a complex aneurysm. The artery can then be spared either by fashioning a horseshoe-shaped skin incision or by identifying its course using Doppler ultrasound prior to skin incision and then cutting down on the artery and following it until sufficient length is provided. The muscle layers are split vertically en bloc and elevated subperiosteally off the bone. Care must be taken not to injure the extracranial VA close to the foramen magnum. The VA can be challenging to palpate and can be identified with the help of Doppler ultrasound. The scalp and muscles are then held apart with self-retaining retractors such as Adson cerebellar retractors, one inserted from the cranial end and a second either overlapping this and centered lower in the incision or inserted from the caudal end.

An osteoclastic craniectomy is preferred because even if a flap has been cut out first, substantial additional drilling laterally will be necessary (▶ Fig. 15.1). The opening is extended superolaterally. The mastoid emissary vein emerges from the mastoid part of the temporal bone and generally indicates the location of the underlying lateral sinus at the junction with the sigmoid sinus. Opened mastoid cells are sealed immediately with bone wax. Larger cells may require plugging with muscle. The craniectomy should measure at the end some 3 to 4 cm in diameter. Caudally, the craniectomy should reach the floor of the posterior fossa. It is usually not necessary to do work around the extracranial segment of the VA, and it is not necessary to remove any part of the atlas.

The dura is opened in a **Y**-shaped fashion for exposure of the caudal cerebellopontine angle and CSF is drained primarily from the cisterna magna for brain relaxation. Next, the ipsilateral cerebellar tonsil is slightly elevated with a retractor and the proximal VA and cranial nerves IX to XI are identified. The VA is located medial and rostral to the dentate ligament; the origin of the PICA is usually located between the fascicles of the cranial nerves IX through XII, but it varies substantially.[4] If there is a distal PICA origin, i.e., close to vertebrobasilar junction, PICA aneurysms are generally located superior to the hypoglossal nerve (suprahypoglossal triangle), whereas with a more proximal PICA origin, i.e., close to the dural entry point of the VA, PICA aneurysms may be found within a triangle between the vagal and accessory nerve (vagoaccessory triangle) or inferior to the hypoglossal nerve (infrahypoglossal triangle).[4,8] Fenestrated or slightly curved aneurysm clips can be useful in order to avoid injury to the cranial nerves (▶ Fig. 15.2). Following clip application and verification of complete occlusion of the aneurysm and patency of the PICA by Doppler sonography and indocyanine green videoangiography, it is important to pay attention to watertight closure of the dura. Dural leaks are a complication that can result in leakage through the skin and infection, space-occupying pseudomeningocele, possibly followed by hydrocephalus or even cervical syringomyelia. The craniotomy defect is handled by replacing the bone with methyl methacrylate as an additional means to prevent a pseudomeningocele. The muscles and fascia are closed in layers and the skin is closed according to surgeon preference.

15.4.2 Midline Suboccipital Approach

For the midline suboccipital approach, the park bench position is favored. A midline skin incision is made from above the external occipital protuberance to the spinous process of the axis (▶ Fig. 15.1). Electrocautery is used to split the muscles in the avascular plane of the ligamentum nuchae. The occipital bone is exposed subperiosteally from the superior nuchal line to the foramen magnum. After placing initial burr holes over the upper lateral edges of the exposure, a free or osteoclastic craniotomy is performed. The dura is opened by making a Y incision down over both hemispheres and then caudally across the foramen magnum. The marginal sinus can be suture ligated or coagulated; the latter will shrink the dura and usually necessitate an autologous or other duroplasty. Similarly, the inferior occipital sinus may have to be suture ligated or coagulated.

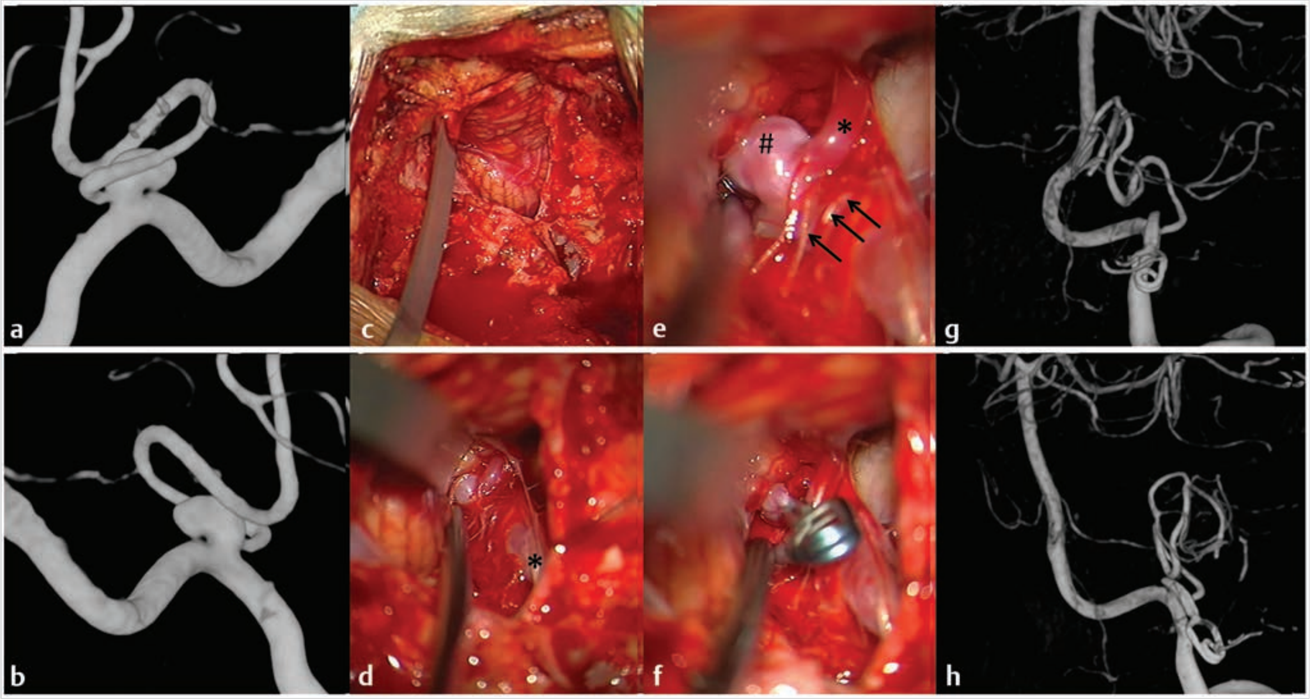

Fig. 15.2 Illustrative case of a ruptured, proximal PICA aneurysm undergoing microsurgical repair. **(a, b)** Preoperative catheter angiography showing a saccular proximal PICA aneurysm with the PICA arising from the aneurysm neck. **(c)** Intraoperative view of cerebellar hemisphere exposition following a midline suboccipital approach, craniectomy, and dura opening. **(d)** After gentle retraction of the cerebellar tonsils, the proximal vertebral artery (*) is exposed. **(e)** Distal dissection along the vertebral artery exposes the PICA (*) and the aneurysm (#), cranial and rostral to the fascicles of the vagal nerve (*arrows*). **(f)** The aneurysm is ultimately clipped using a curved clip. **(g, h)** Postoperative catheter angiography confirms the complete aneurysm occlusion and patency of the PICA.

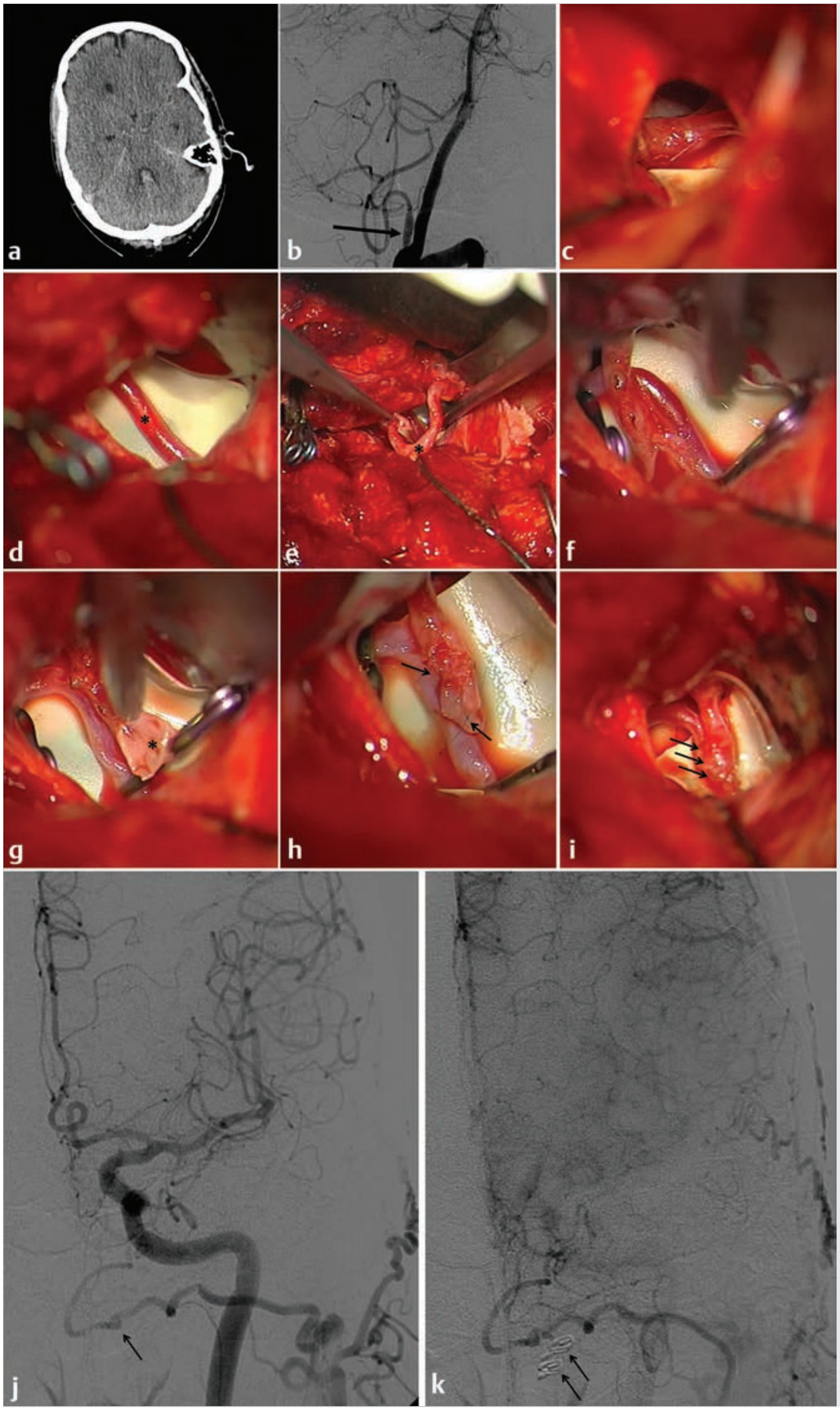

Fig. 15.3 **(a)** CT scan demonstrating an isolated fourth ventricular hemorrhage. **(b)** Catheter angiography confirms the presence of a fusiform PICA aneurysm, which was not amenable for endovascular repair without sacrifice of the parent artery. The interdisciplinary decision-making yielded surgical treatment, i.e., occipital artery-to-PICA bypass and trapping of the aneurysm. **(c)** Intraoperative view after exposition of the aneurysm. After preparation and exposure of the distal segment of the PICA **(d, *)** and the occipital artery **(e,*)**, both vessels are approximated **(f)**. Following temporary clipping (*arrows*) of the recipient vessel, the arteriotomy—matching the fish-mouthed donor vessel (*)—is made **(g)**. Then positional stiches are placed **(h,** *arrows***)** at both ends of the arteriotomy, following stiches that are placed along the back and then front wall in either continuous or discontinuous way **(i,** *arrows***)**. **(j, k)** Postoperative catheter angiography demonstrates the patent occipital artery to distal PICA bypass, with retrograde filling of the PICA (**j**, *arrow*) as well as the complete trapping of the fusiform PICA aneurysm (**k**, *arrows*).

Next, after CSF drainage from the cisterna magna and gentle elevation of the cerebellar tonsils, the distal PICA is identified and then followed proximally or distally to the aneurysm (▶ Fig. 15.3). Depending on its location, the aneurysm is then identified and eventually clipped. However, distal PICA aneurysms are generally broad based or fusiform, and the maintenance of parent vessel integrity with clipping is not always possible. In the case of a peripheral aneurysm, trapping with occlusion of the parent artery usually can be done without negative consequences, but in case of a dominant PICA supplying a large territory, preservation of vascular continuity is mandatory. Reconstructive measures must be planned; options include end-to-end anastomosis after resection of the aneurysm, PICA–PICA side-to-side anastomosis, or occipital artery-to-PICA bypass (▶ Fig. 15.3). The dura is closed in watertight fashion with an autologous pericranial graft if necessary. The bone is replaced with methyl methacrylate and the incision closed in routine fashion.

15.5 Postoperative Management Including Possible Complications

Patients undergoing posterior fossa surgery should be monitored in an intensive care unit setting postoperatively. Any neurological change, altered consciousness, or substantial, persistent change in vital signs such as respiration should be investigated with a CT scan to rule out a posterior fossa hematoma or acute obstructive hydrocephalus. A functioning ventricular drain will assist with ruling out the latter. If there is no ventricular drain in place and the patient deteriorates acutely with decreasing level of consciousness and respiratory compromise or arrest such that intubation is required, a CT scan should be obtained immediately and emergency placement of a ventricular drain considered. Other care is the same as with other SAH patients, including adequate fluid replacement to maintain normovolemia, avoidance of factors that adversely affect brain perfusion or that increase brain metabolic rate, nimodipine, mobilization, venous thromboembolism prophylaxis, and monitoring clinically and by transcranial Doppler ultrasound, CT perfusion, or some other method for clinical and radiological signs of delayed cerebral ischemia.

The main specific possible complications include either or both brainstem and cranial nerve injury, vascular injury to the transverse or sigmoid sinus or either major arteries or small perforators with attendant ischemic injury, postoperative hematoma, CSF leakage, and cerebellar injury from retraction. Lower cranial nerve palsies can lead to dysphagia and vocal cord paralysis, so the patient needs a careful swallowing evaluation postoperatively before being allowed to eat and drink.

The airway should be deemed adequate before extubation. A gastrostomy tube and tracheostomy are needed occasionally if there is severe dysfunction. Neurological monitoring is done for several days because brain swelling can develop over this time. A postoperative catheter angiography may be done to confirm aneurysm obliteration and patency of major arteries.

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16 Retrolabyrinthine Transsigmoid and Extreme Lateral Infrajugular Transcondylar-Transtubercular Exposures for Aneurysms

Jonathan J. Russin, Alexandra Kammen, and Steven L. Giannotta

Abstract
This chapter focuses on the retrolabyrinthine transsigmoid (RTL) and extreme lateral infrajugular transcondylar-transtubercular exposure (ELITE). These skull base approaches are highlighted for the treatment of intracranial aneurysms. The shared benefit of these exposures is a more anterior view of the posterior fossa while limiting the need to mobilize the brainstem. The RTL provides access farther along the vertebrobasilar system. The ELITE favors a more caudal exposure. These complex skull base techniques are mainly applied in conjunction with endovascular therapies or in case no effective endovascular treatment is available.

Keywords: retrolabyrinthine transsigmoid, extreme lateral infrajugular transcondylar-transtubercular exposure, aneurysm, posterior circulation, skull base approach

16.1 Introduction

Aneurysms of the posterior circulation represent a unique surgical challenge due to their limited accessibility and worse natural history compared to anterior circulation aneurysms.[1] The popularization of skull base approaches has improved the outcomes for patients presenting with these lesions. More recently, advances in endovascular technology have provided less invasive treatment options for these lesions, reducing the need for open surgical intervention. This paradigm has benefitted patients but has also resulted in some of the most difficult cases being selected out for surgical treatment. The skull base techniques described here are well established for the treatment of posterior circulation aneurysms. Applying these approaches in conjunction with endovascular therapies has the potential to help create new paradigms that maximize patient safety and therapeutic success.

16.2 Patient Selection

The retrolabyrinthine transsigmoid (RLT) and extreme lateral infrajugular transcondylar-transtubercular exposure (ELITE) are indicated for select aneurysms of the basilar artery—anterior inferior cerebellar artery (AICA) junction, lower basilar trunk, vertebrobasilar junction, the distal vertebral artery, and proximal posterior inferior cerebellar artery (PICA) (▶ Fig. 16.1). Aneurysms more distal on the AICA and PICA typically do not require the anterior exposure afforded by these approaches. Additionally, the geometry, orientation, and size of the aneurysm must be considered when planning the surgical approach. These approaches can be particularly useful for aneurysms that project posteriorly into the basilar sulcus because of the more anterior line of approach that can be achieved.

The laterality of approach is governed by the geometry of the aneurysm, the anatomical orientation of its neck, the relative sizes of the sigmoid sinuses when using the RLT, the collateral venous outflow pattern, and the presence of an internal jugular vein on the ipsilateral side as assessed by preoperative venous phase angiography or computed tomography venography. Most vertebrobasilar junction aneurysms and basilar artery trunk aneurysms point either ventrally or dorsally, and can be approached on the side of the smaller sigmoid sinus. If the origin of the neck is strongly lateralized to one side or the basilar artery

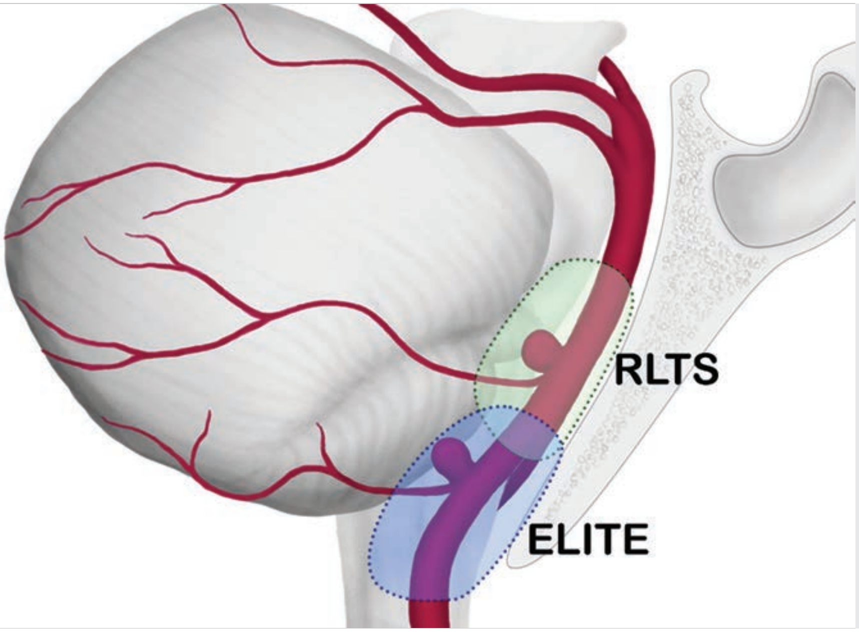

Fig. 16.1 The exposures for both the ELITE and RLT approaches.

is deviated to one side, the approach should be made ipsilateral to that side. For aneurysms directed superiorly or inferiorly, the approach is made on the side of the smaller sigmoid sinus.

Application of these approaches should be limited to those lesions that reside anterior to the brainstem. Lateralized lesions can typically be approached using retrosigmoid or far lateral exposures that reduce the risk to neurovascular structures. These approaches are also recommended only in conjunction with endovascular therapies or in case no effective endovascular therapy is available.

16.3 Contraindications

The RLT approach is not recommended for patients with a diminutive or absent contralateral sigmoid sinus. The ELITE is relatively contraindicated when there is instability at the craniocervical junction because bony removal of the condyle could further exacerbate instability.

16.4 Preoperative Planning

When planning to ligate the sigmoid sinus for the RLT approach, consideration can be given to preoperative testing. Temporary occlusion of the sinus with measurement of the venous sinus pressure proximal to the occlusion can be used. A rise in sinus pressure of less than 5 cm H_2O likely conveys a reasonable margin of safety for sinus ligature.[2] This measurement can also be made intraoperatively. However, in the senior author's experience, the most important factor is the presence of a satisfactory contralateral sigmoid sinus on imaging. It is no longer our practice to perform preoperative or intraoperative venous pressure measurements.

Careful review of preoperative imaging, in particular the bony anatomy of the occipital condyle and the jugular tubercle, can assist the surgeon in effective and safe performance of the ELITE approach. Measurements of the tubercle as well as relationships to the hypoglossal canal and jugular bulb can help facilitate aneurysm exposure while minimizing the required bony removal.

When managing aneurysmal subarachnoid hemorrhage, routine cerebrospinal fluid (CSF) diversion is planned for both the RLT and ELITE procedures. Elective cases are considered individually for possible CSF diversion based on the location of the lesion and the cisternal space available for dissection. A ventriculostomy is often placed for those patients who experience aneurysmal subarachnoid hemorrhage, while a lumbar drain is typically chosen for patients being treated electively. CSF diversion is used to limit the need for retraction and may be continued postoperatively to facilitate wound healing.

16.5 Operative Procedures

16.5.1 Retrolabyrinthine Transsigmoid

Patients are positioned either supine with the head angled 45 degrees away from the side of entry or in a park bench position. It is our preference to position supine when the procedure is contralateral to the handedness of the surgeon and in park bench when the procedure is ipsilateral to the handedness of the surgeon. Pin headrest fixation is not required; however, it is our practice to use a Mayfield skull clamp in almost all cases. This helps ensure a stable surgical field and supports self-retaining retractors. Facial nerve electromyography, brainstem auditory evoked responses, and motor and sensory evoked responses are monitored routinely.

A curvilinear retroauricular scalp incision is made approximately 3 cm behind the ear crease, extending from the asterion or just above the nuchal line to just below the rim of the foramen magnum (▶ Fig. 16.2). The skin and subcutaneous tissue are elevated in a separate layer from the muscle and fascia. Additionally,

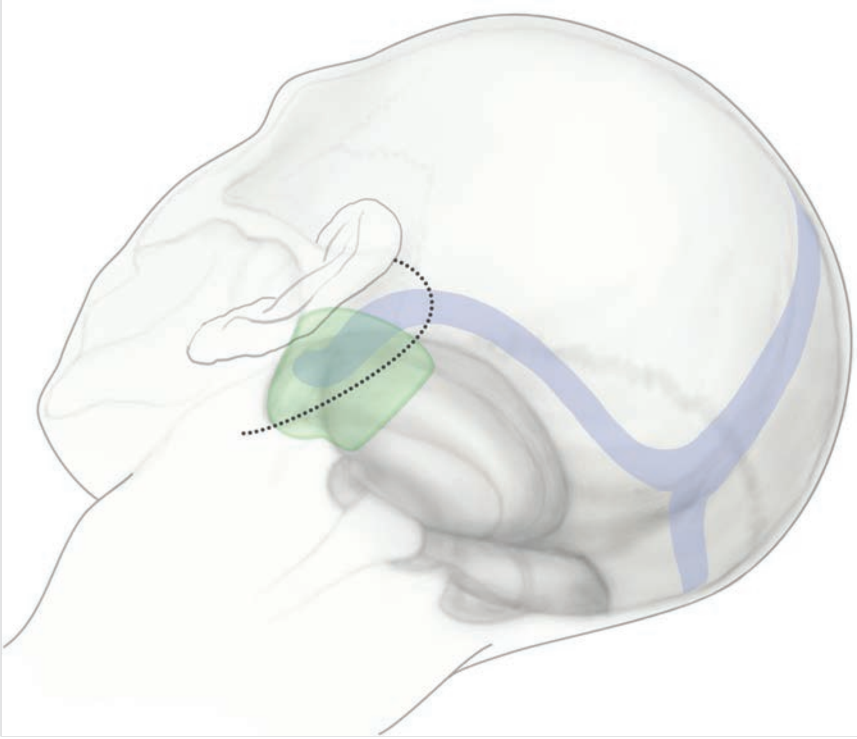

Fig. 16.2 The location of the RLT incision as well as the extent of the craniotomy in relationship to the sigmoid sinus.

the fascial incision is offset slightly from the skin incision. This facilitates a multilayer closure with offset suture lines to help prevent postoperative CSF leaks. The musculocutaneous flap is then reflected anterolaterally with fishhooks to expose the posterior lip of the external auditory meatus and the spine of Henle. A modified mastoidectomy with retrolabyrinthine exposure is performed using a cutting or diamond burr on a high-speed drill. The posterior semicircular canal is skeletonized and the posterior fossa dura, anterior to the sigmoid sinus (Trautmann's triangle), is exposed. The sigmoid sinus, from its junction with the superior petrosal sinus to its junction with the jugular bulb, is egg-shelled and the posterior fossa dura for approximately 3 to 4 cm behind the sigmoid sinus is exposed. The limits of the mastoidectomy include the floor of the middle fossa and superior petrosal sinus superiorly, the jugular bulb inferiorly, the posterior semicircular canal anteriorly, and the retrosigmoid dura posteriorly. If there is preexisting hearing loss on the side of the approach, this enables removal of the labyrinth in order to widen the exposure toward the clivus.

The retrosigmoid dura is then opened sharply and reflected up against the posterior margin of the sigmoid sinus (▶ Fig. 16.3). It has been our practice to sacrifice the sigmoid sinus if preoperative imaging showed a patent and comparable contralateral sinus. When doubt exists about the size of the contralateral transverse and sigmoid sinus, then a temporary occlusion of the ipsilateral sigmoid can be performed and a small-gauge needle can be used to measure the change in pressure in the ipsilateral transverse sinus. If the sinus pressure rises less than 5 cm H_2O, sinus ligation is reasonably safe. In the case of an elevation in pressure greater than 5 cm H_2O, the presigmoid dura can be opened and surgical corridors are available retro- and presigmoid. Alternatively, the superior petrosal sinus can be divided and the craniotomy can be extended slightly superiorly to create a transpetrosal exposure.

Once the sigmoid sinus is ligated and divided at both ends, the entire dural flap can be reflected anteriorly. The center of the operative field typically includes the cochlear–facial nerve complex and the flocculus of the cerebellum. The fifth cranial nerve is present superiorly and deep in the exposure, spanning the subarachnoid space until its entrance into Meckel's cave. Inferiorly, the 9th, 10th, and 11th cranial nerves are in view as they enter the jugular foramen (▶ Fig. 16.4). Immediately after the dura is opened, the cisterna magna is opened, which allows for further relaxation of the cerebellar hemisphere. The arachnoid of the prepontine and cerebellopontine angle cisterns is then opened to expose the vertebral artery. Dissection can be carried along the ipsilateral vertebral artery, working in windows between the cranial nerve complexes identified above, to the vertebral-basilar junction. The basilar artery can typically be followed superiorly as far as the origin of the AICA. In some cases, the exposure can reach as far cranially as the origin of the superior cerebellar artery. At this point, proximal and distal exposures of the ipsilateral vertebral artery, the basilar artery, the PICA, and the AICA are accessible, facilitating control of these arteries in preparation for clip ligation. Secondary to the narrow corridors between the cranial nerves, low-profile clip appliers are optimal. Despite the use of these specialized appliers, it is frequently the case that the head of the clip applier will either obscure the surgical view or be too large for the surgical corridor. As an alternative, long aneurysm clips can be used to help remove the head of the applier from the surgical view and allow application without passing the applier deep within the exposure.

Following clip ligation, a watertight dural closure is performed. Frequently, this is not possible and a suturable dural substitute is used to recreate a dural seal. It is our practice to place an oversized piece of the suturable dural graft intradurally and then to close the defect by passing our needle through the dural flap, the suturable graft, and then the dural edge to create a sandwich effect. This allows the CSF pulsations to adhere the graft to the durotomy. Routinely, autologous abdominal fat grafts are used to fill in the dead space created by the mastoidectomy and prevent CSF leaks into the mastoid air cells. Titanium skull base plating is used to reconstruct the craniectomy. Although we do not place lumbar drains for CSF diversion in all cases, they are always an option for prevention when dural reconstruction is challenging or potentially incomplete (**Video 16.1**).

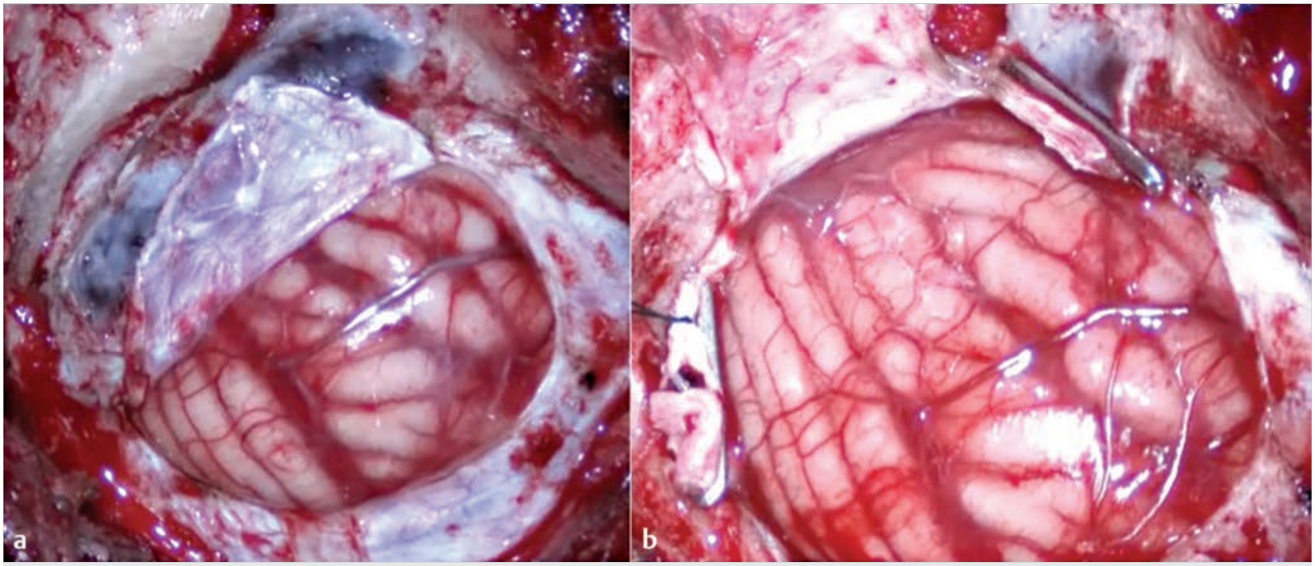

Fig. 16.3 (a) Intraoperative photo showing exposure prior to ligation and division of the sigmoid sinus. **(b)** RLT exposure in the case of a ruptured basilar trunk aneurysm. Extensive subarachnoid hemorrhage is appreciated over the cerebellar hemisphere.

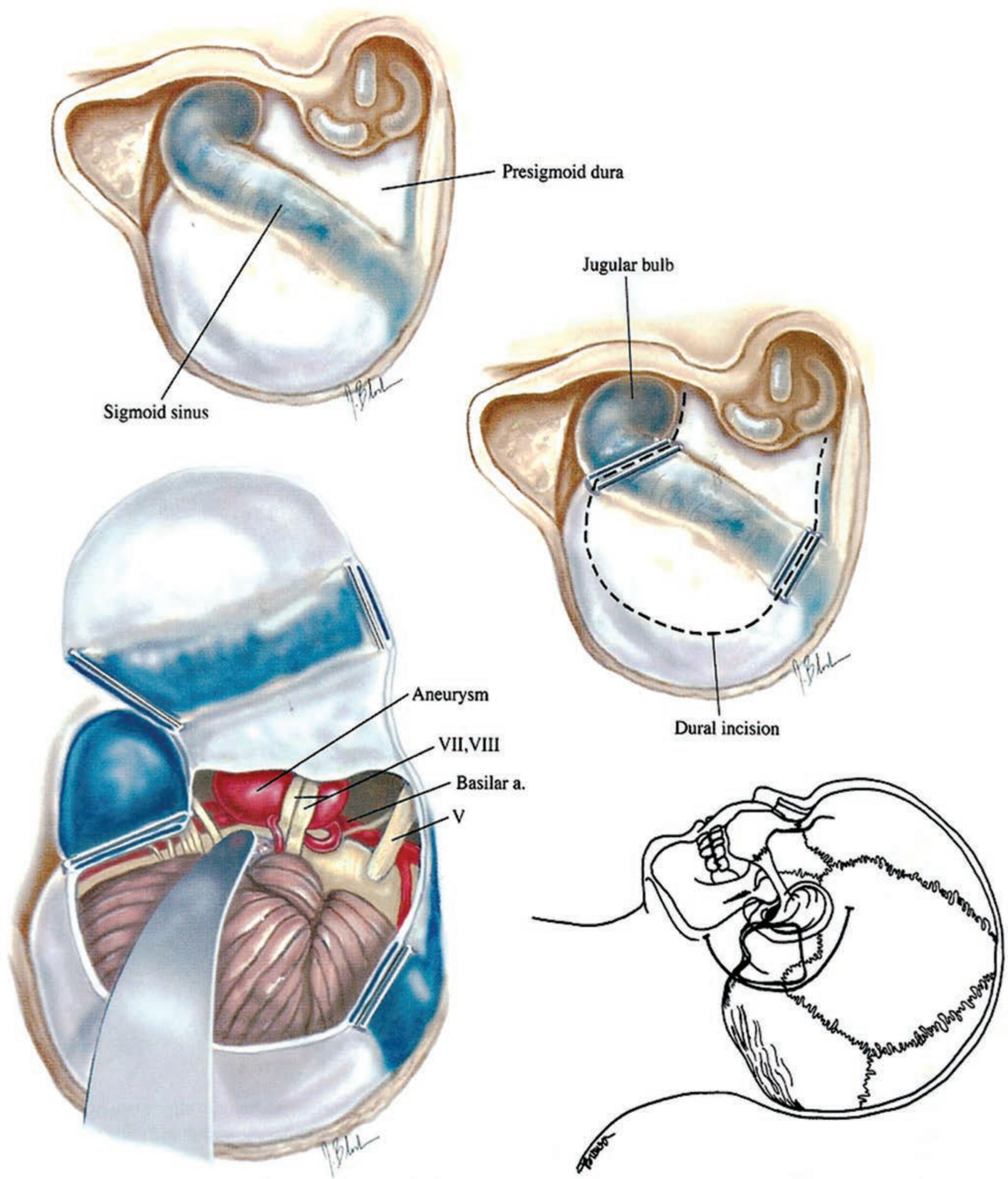

Fig. 16.4 A complete mastoidectomy with skeletonization of the presigmoid and retrosigmoid dura in the RLT approach (*upper left*). The posterior semicircular canal is completely skeletonized. The sigmoid sinus is then ligated superiorly and inferiorly as part of the dural opening (*center*). A wide retrolabyrinthine exposure of the vertebrobasilar junction area is afforded. The aneurysm is exposed behind the facial–auditory complex (*lower left*). Inset at lower right shows the retroauricular scalp incision and craniotomy for a left-sided RLT approach. (Reproduced with permission from Giannotta SL, Farin A. Retrolabyrinthine transsigmoid and extreme lateral inferior transcondylar exposures for aneurysms. In: Macdonald RL, ed. Neurosurgical Operative Atlas: Vascular Neurosurgery. 2nd ed. New York, NY: Thieme; 2009:88.)

16.5.2 Extreme Lateral Infrajugular Transcondylar-Transtubercular Exposure

Positioning is typically in the three-quarter lateral decubitus or park bench position with the patient's head in three-point pin fixation. The vertex is oriented slightly downward to make the patient's ear the highest point in the field. The shoulder is positioned out of the surgical field by moving it caudally, rotating it anteriorly, and fixing it with adhesive tape. Care is taken not to pull excessively on the shoulder to avoid injury to the brachial plexus. A vertical "lazy S" or linear retromastoid incision is made starting 2 cm posterior to the mastoid bone at the level of the external auditory meatus and extending caudad along the posterior body of the mastoid and the posterior aspect of the sternocleidomastoid muscle to the level of approximately C2/C3. The superficial muscular layer, consisting of the sternocleidomastoid and splenius capitis muscles laterally and the trapezius and semispinalis capitis muscles medially, is identified and divided to allow for exposure of the deep fascia and longissimus capitis muscle. Dividing the longissimus capitis and opening the deep fascia allows visualization of the suboccipital triangle. This muscular triangle is bordered by the superior oblique, inferior oblique, and rectus capitis major muscles. The arch of C1, the vertebral artery, and the C1 nerve root can be found in the suboccipital triangle.

After reflecting the musculature of the suboccipital triangle, the vertebral artery is palpated as it arches over the atlas (C1) to pierce the atlanto-occipital membrane. The course of the vertebral artery is variable, and careful review of preoperative imaging is a requisite for safe dissection. In the case that mobilization of the vertebral artery is required, the soft tissue and venous plexus around the extradural portion can be coagulated and removed. A posterior meningeal artery typically originates just proximal to the dural entrance of the vertebral artery and can be divided if necessary. However, the posterior spinal artery can originate at or near the dural ring, and care should be taken to preserve it. Additionally, an extradural origin of the PICA is not unusual and should also be considered on preoperative imaging.

A retromastoid craniectomy is then performed and extended through the foramen magnum. A high-speed drill is used to remove the rim of the foramen magnum as well as the inferior portion of the mastoid to expose and skeletonize the distal sigmoid sinus and jugular bulb. In the event that more inferior exposure is required, the posterior arch of the atlas can be removed from the midline to the lateral mass. A hemilaminectomy of the axis (C2) can also be performed, and, if necessary, the vertebral artery can be transposed out of the transverse foramen. After extending the craniectomy to the posterior margin of the sigmoid sinus and jugular bulb, the posterior one-third of the occipital condyle is removed, until the hypoglossal canal and jugular tubercle are exposed. Drilling can continue anteromedially and superiorly to expose the undersurface of the jugular bulb. The jugular tubercle can be reduced by drilling between the hypoglossal canal and the jugular bulb. The hypoglossal nerve can be skeletonized in its canal to facilitate anterior exposure (▶ Fig. 16.5).

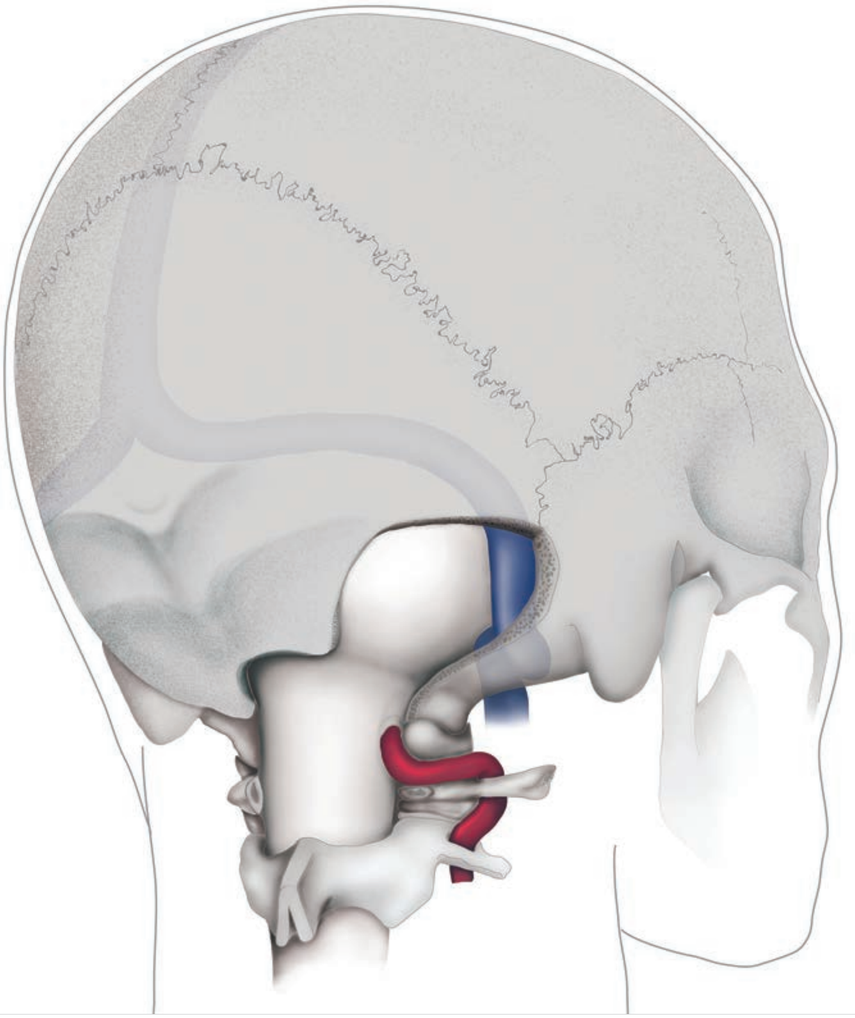

Fig. 16.5 The skull base craniotomy for the ELITE approach.

Once the skull base drilling is complete, the dura is incised vertically, beginning at the transverse–sigmoid sinus junction paralleling the entire length of the exposed sigmoid sinus, posterior to the vertebral artery dural entrance, and extending as far inferior as needed. The dura is reflected anterolaterally, carrying with it the inferior portion of the sigmoid sinus. Several variations of dural opening can be used to facilitate accessing particular pathology. The exposure provides an inferior–superior trajectory combined with a lateral–medial trajectory to the lower clivus and vertebrobasilar complex, such that the line of sight is parallel with the vertebral artery anterior to the lower brainstem. The exposure is best suited for lesions involving the vertebrobasilar junction and lower basilar artery trunk (▶ Fig. 16.6). The need for brainstem retraction is minimized with the ELITE approach, while facilitating proximal control of relevant vasculature.

Fusiform aneurysms of the distal vertebral artery, most of which are dissections, are best treated with trapping. However, the frequent involvement of the PICA origin puts brainstem perforators from the anterior and lateral medullary segments of the PICA at risk. Combining open surgical and endovascular techniques can prove optimal for these complex aneurysms (▶ Fig. 16.7).

Dural reconstruction is important to prevent postoperative CSF leaks or pseudomeningocele formation. Frequently, primary watertight dural closure is not possible. It has been our practice to use a suturable dural substitute placed intradurally, beyond the edges of the durotomy, to reconstruct a watertight seal. Bone wax and/or fibrin glue is frequently used to seal off the mastoid air cells. High-risk cases can be managed with an autologous fat graft. A titanium mesh can be used to reconstruct the craniectomy. Meticulous fascial closure is also critical for ideal wound healing. Although it is not our routine practice, CSF diversion for a few days postoperatively can be useful to prevent CSF leaks in select patients.

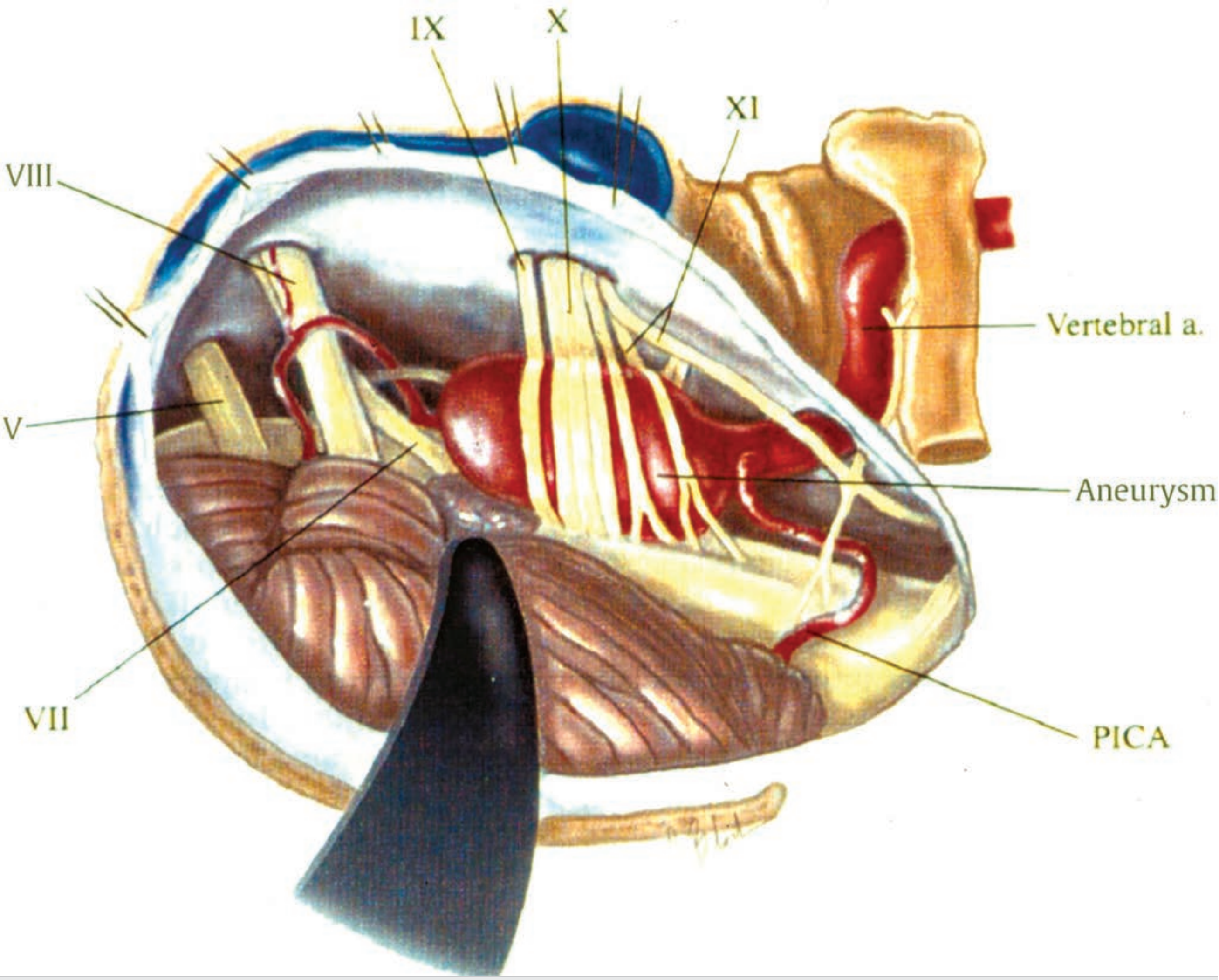

Fig. 16.6 The ELITE approach provides wide exposure anterior to the brainstem with only a minimum of retraction. The relevant cranial nerves and vascular structures are labeled. (Reproduced with permission from Giannotta SL, Farin A. Retrolabyrinthine transsigmoid and extreme lateral inferior transcondylar exposures for aneurysms. In: Macdonald RL, ed. Neurosurgical Operative Atlas: Vascular Neurosurgery. 2nd ed. New York, NY: Thieme; 2009:91.)

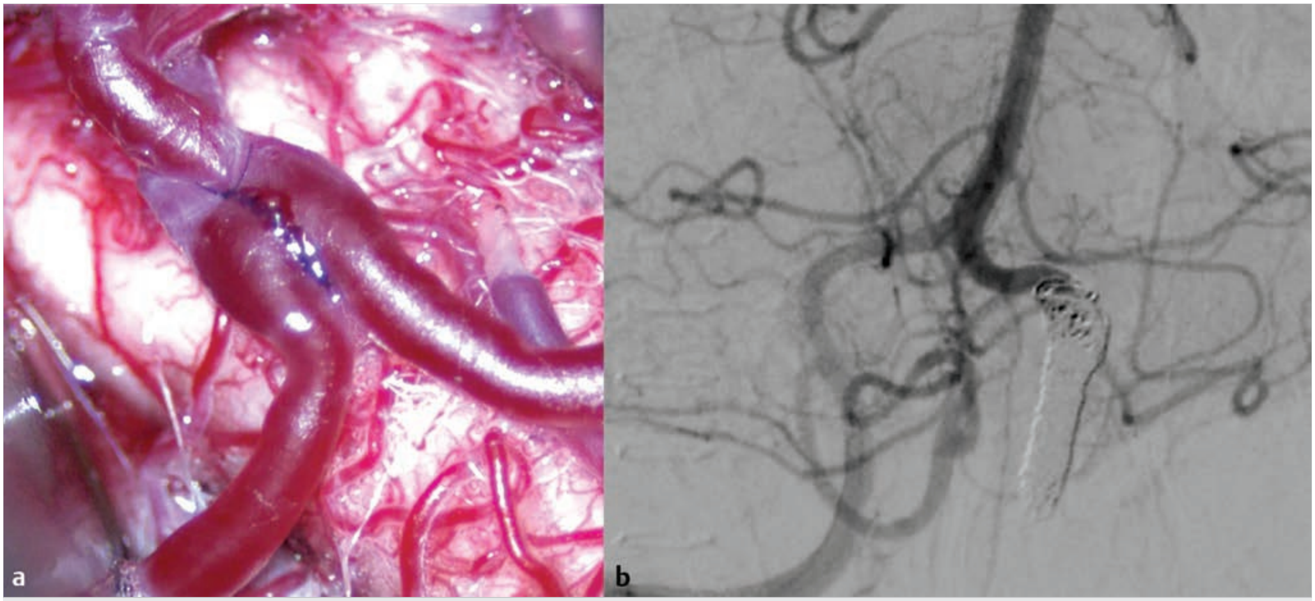

Fig. 16.7 **(a)** Intraoperative picture of a completed PICA–PICA bypass. This was performed prior to the endovascular embolization of a fusiform vertebral artery aneurysm that presented with subarachnoid hemorrhage. **(b)** Anteroposterior angiogram after endovascular sacrifice of the fusiform aneurysm of the vertebral artery. Filling of the bypass is appreciated with robust distal filling bilaterally and a small amount of flow seen filling retrograde to the brainstem perforators on the side of the endovascular occlusion.

16.6 Postoperative Management Including Possible Complications

Patients are managed postoperatively in an intensive care setting. Complications of the retrolabyrinthine, transsigmoid approach include transient or permanent hearing loss and injury to the lower cranial nerves that may result in vocal cord paralysis, dysphagia, and aspiration pneumonia. Tracheostomy and gastrostomy may be necessary in some cases of severe persistent cranial neuropathy. The facial nerve is also vulnerable to injury. Treatment acutely focuses on protecting the cornea, maintaining corneal lubrication, and possibly tarsorrhaphy, if recovery is not prompt. Delayed procedures for reconstruction of facial nerve function can be considered if the nerve is completely lost. A more common complication is CSF leak requiring diversion or surgical revision. Specific to the transsigmoid approach is the risk of venous infarction.

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17 Vertebral Confluence and Midbasilar Aneurysms Including Transpetrosal Approach

Hasan A. Zaidi, Vini G. Khurana, Douglas John Fox Jr., L. Fernando Gonzalez, and Robert F. Spetzler

Abstract
Vertebral confluence and midbasilar aneurysms are uncommon but surgically challenging lesions due in large part to the ventral location of pathology in the posterior fossa and the important downstream territory this vascular tree supplies. Four general open surgical approaches are used to treat these lesions: the orbitozygomatic/subtemporal approach, the extended middle fossa (Kawase)/transpetrosal approach, the retrosigmoid approach, and the far-lateral approach. Each provides access to different segments of the posterior circulation. In this chapter, we review important surgical and clinical pearls in the treatment of these lesions.

Keywords: aneurysms, basilar artery, clipping, transpetrosal, vertebral artery

17.1 Introduction

Vertebral confluence and midbasilar aneurysms are uncommon but surgically challenging lesions due in large part to the ventral location of pathology in the posterior fossa and the important territory this vascular tree supplies. Surgical principles paramount to successful surgical obliteration of these lesions include careful analysis of preoperative vascular imaging to delineate location of critical brainstem perforating vessels, maximizing surgical visualization of the lesion with extensive bony resection and dural opening, careful and early dissection of proximal and distal vessels, preservation of all vascular perforators, and development of contingency plans for use when primary clip occlusion is not feasible.

17.2 Patient Selection

17.2.1 Diagnosis

Vertebral confluence and midbasilar aneurysms are defined by their location anywhere along the lower three-fifths of the basilar artery. This includes lesions from the vertebrobasilar junction into the origin of the anterior inferior cerebellar artery (AICA). The gold standard in diagnosis remains formal, four-vessel angiography in order to define the morphology of the aneurysm as well as to delineate the relationship of important perforating vessels to the aneurysm dome. However, perforators may not always be immediately apparent and are often obscured when treating giant aneurysms. Furthermore, injections of the internal and external carotid and vertebral arteries and three-dimensional rotational views can help determine the caliber of vessels and visualize the posterior communicating arteries in order to develop contingency plans for use when primary clipping may not be feasible and bypass may be necessary. Computed tomography (CT), CT angiography, and magnetic resonance imaging including magnetic resonance angiography are important supplementary studies that can be used during intraoperative neuronavigation, to determine the degree of bony opening and surgical approach during presurgical planning, as well as to detect the presence of calcifications and thrombus in the aneurysm. However, these studies should not obviate or replace the formal angiogram that can provide much greater anatomical detail.

17.2.2 Indications/Contraindications

Surgical indications include lesions not amenable to neuroendovascular treatment, including wide-necked aneurysms, and aneurysms with important brainstem perforators within the aneurysm dome among patients with the potential for excellent functional recovery. The risk of surgical morbidity from treating unruptured lesions in functionally asymptomatic patients needs to be weighed against the risk of the natural history of these lesions. The risk of rupture causing irreversible morbidity is directly related to the size of the aneurysm dome, irregular appearance of the aneurysm and parent vessel, and the patient's personal or family history of aneurysmal subarachnoid hemorrhage. Contraindications for open surgical treatment include lesions in patients with extensive comorbid medical conditions that would place them at higher risk of medical complications from prolonged anesthesia or blood loss. Patients with extensive cardiopulmonary or airway issues as well as advanced age should be counseled on the extensive risk of surgery, and should proceed only if the risk of the natural history outweighs the risk of surgical treatment.

17.2.3 Timing, Alternatives, and Risks

Like many ruptured aneurysms, the risk of rerupture is greatest in the first 24 to 48 hours after the initial event, and surgical obliteration procedures should be performed early in these patients. Patients with unruptured aneurysms should be treated after a comprehensive medical and radiological workup is completed, including an assessment of the risk of medical complications with cardiac standstill procedures. The main alternatives are close radiological observation or endovascular treatment, which requires input from an endovascular neurosurgeon or neurointerventional radiologist.

17.2.4 Surgical Approaches and Anatomical Considerations for Each Approach

There are four general approaches to the treatment of vertebral confluence and midbasilar aneurysms: the orbitozygomatic/subtemporal approach, the extended middle fossa (Kawase)/transpetrosal approach, the retrosigmoid approach, and the far-lateral approach. The orbitozygomatic and subtemporal approaches allow access to the upper two-fifths of the basilar artery and are generally appropriate for treating aneurysms located at the basilar bifurcation, precommunicating segment of the posterior cerebral artery, or superior cerebellar artery. The extended middle fossa (Kawase) and transpetrosal approaches are appropriate for lesions located on the middle fifth of the basilar artery, typically AICA lesions. The retrosigmoid approach is used for aneurysms of the vertebrobasilar junction up to the AICA (lower three-fifths of the basilar artery), and the far-lateral approach is used for lesions of the vertebral artery from its intradural origin to the vertebrobasilar junction. In our experience, we have found that the orbitozygomatic, retrosigmoid, and far-lateral approaches generally provide plentiful visualization of the majority of lesions.

17.3 Preoperative Preparation

Due to the potential for high-volume blood loss, we routinely place central venous lines and use continuous blood pressure monitoring with arterial lines during surgery for aneurysms. The patient is typically typed for blood group and screened prior to surgery. If cardiac standstill is planned, a Swan-Ganz catheter is placed. Perioperative antibiotics are administered. Electroencephalography and somatosensory evoked potential (SSEP) monitoring are routinely used for both surgical positioning with neck rotation and intraoperative monitoring for adequate blood flow after clip reconstruction. Cranial nerve monitoring is not routinely used, but may include seventh and/or eighth nerve monitoring of brainstem auditory evoked potentials in order to monitor brainstem function. Motor evoked potentials are used for patients with aneurysms involving the basilar apex and for cardiac standstill cases.

Prior to performing the craniotomy, mannitol (0.5 g/kg) is given in most cases. The goals for blood pressure and $PaCO_2$ are normotension and normocapnia during the majority of the procedure. After temporary occlusion, the blood pressure may be elevated pharmacologically to promote brain perfusion. If preoperative imaging or intraoperative inspection demonstrates a friable aneurysm neck or dome, the blood pressure can be lowered during dissection to reduce the chances of an intraoperative rupture. Anticonvulsants are seldom used. Common to all the approaches described here are the use of lumbar drainage for up to 72 hours postoperatively and preparation and draping of the abdomen and groin in case fat grafts and intraoperative angiography are used. Collaboration with a neuro-otologist is generally recommended, not just for transpetrosal approaches, but also for postoperative management of ear, nose, and throat issues.

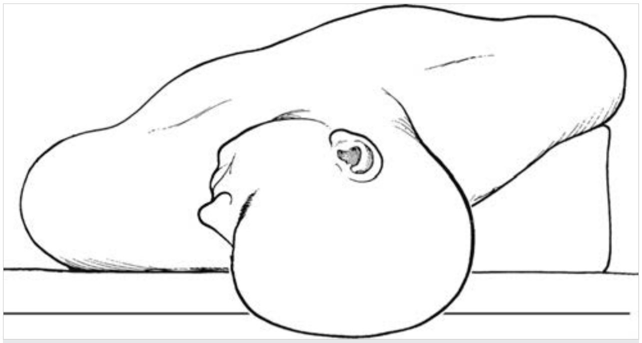

Fig. 17.1 The patient is placed in the supine position for the extended middle fossa approach, with the appropriate shoulder elevated and the head in the horizontal position, slightly extended and tilted 10 to 15 degrees toward the floor. (Reproduced with permission from Barrow Neurological Institute, Phoenix, AZ.)

17.4 Operative Procedures

17.4.1 Extended Middle Fossa (Kawase) Approach

The patient is positioned supine with the head turned 90 degrees contralateral to the surgical approach. Elderly patients with a risk of cervical stenosis should have pre- and postpositioning SSEP monitoring for any change in latency or amplitude. Large shoulder and hip rolls are used as needed to facilitate the head turn and to reduce the degree of neck rotation to promote venous return. The neck is laterally extended and the vertex is tilted 10 to 15 degrees toward the floor to allow the temporal lobe to fall with gravity away from the surgical corridor. To prevent the shoulder from obstructing the surgeon's arms, the shoulder can be taped caudally, but careful attention is paid to avoid excessive traction as this increases the risk of cervical and brachial plexopathy (▶ Fig. 17.1). The head is placed in a three-pin radiolucent head holder in the event an intraoperative angiogram is required.

Either an anteriorly concave question-mark incision or an inferiorly concave horseshoe-shaped incision can be used (▶ Fig. 17.2). The inferior end of the incision immediately anterior to the tragus should reach the inferior margin of the origin

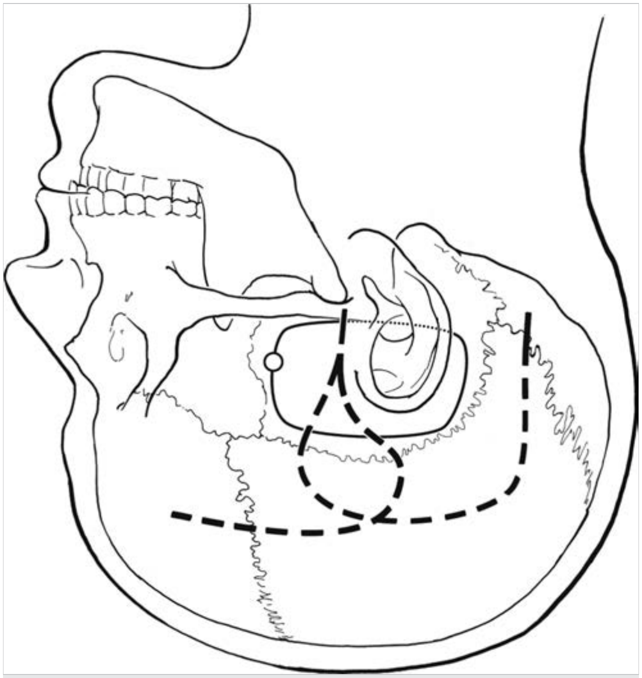

Fig. 17.2 The scalp incision options and craniotomy for the middle fossa approach. It is essential to extend the craniotomy inferiorly until it is flush with the floor of the middle fossa. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:93.)

of the zygomatic process of the temporal bone. The incision should remain less than 1 cm ahead of the tragus to avoid severing the frontalis branch of the facial nerve and ascending trunk of the superficial temporal artery. When a question-mark incision is used, the scalp flap is reflected anteriorly in two layers, in a standard fascia-splitting technique. A zygomatic osteotomy is optional. If one is performed, the authors detach the zygomatic process with two reciprocating saw cuts, keeping it attached to the inferiorly reflected temporalis muscle. For horseshoe-shaped incisions, the scalp flap is reflected inferiorly in one layer.

The temporal bone exposure should be two-thirds anterior and one-third posterior to the external auditory meatus (▶ Fig. 17.3). A temporal craniotomy is performed and the bony opening should be drilled flush with the floor of the middle cranial fossa. An extradural dissection is then carried out from the floor of the middle cranial fossa, starting posteriorly and moving anteriorly. The following structures should be exposed: arcuate eminence, greater superficial petrosal nerve (GSPN) at the facial–geniculate hiatus, middle meningeal artery at the foramen spinosum, and posterolateral margin of the mandibular nerve at the foramen ovale (▶ Fig. 17.4). Continued elevation of the dura medially along the petrous ridge exposes the structures that form the triangle of Glasscock laterally and the triangle of Kawase medially (▶ Fig. 17.5). The bone of the Kawase triangle can then be drilled away carefully using a diamond drill. The petrous segment of the internal carotid artery, which is usually near the inferior aspect of the GSPN, is identified. This drilling exposes the posterior fossa dura. The temporal dura is opened linearly, anteriorly to posteriorly, and careful attention is given to avoid injury to the vein of Labbé. This portion of the dura is then reflected superiorly with tack-up sutures. A second dural incision is extended perpendicularly from the first incision toward the exposed dura of the posterior fossa. A third, posteriorly concave incision is made in the dura of the posterior fossa. The final incision is made through the tentorial incisura, with sacrifice of the superior petrosal sinus as needed. The trochlear nerve is identified and preserved before making the tentorial incision (▶ Fig. 17.6). After the arachnoid has been dissected, the superior cerebellar, posterior cerebral, and midbasilar arteries and cranial nerves III through VIII can be seen.

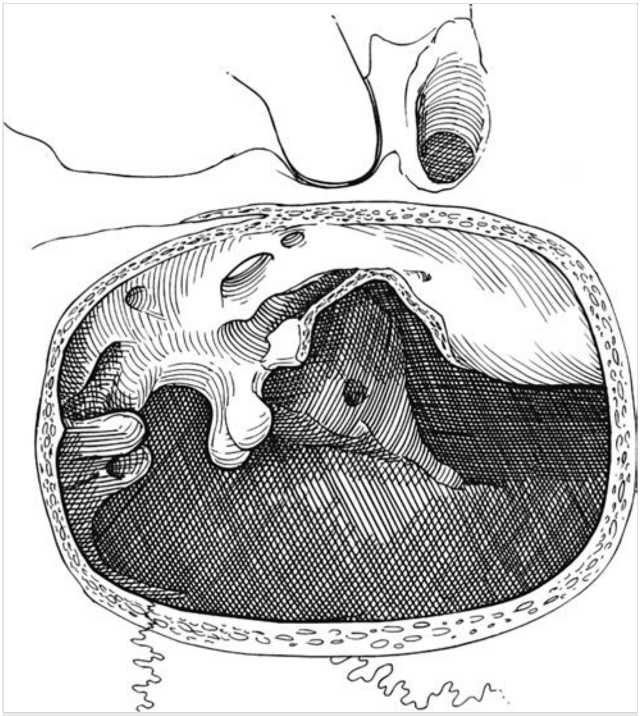

Fig. 17.3 The middle fossa bone exposure is shown. The posterolateral to anteromedial areas of the foramen spinosum, foramen ovale, and foramen rotundum are seen. The foramen lacerum is medial and just lateral to the posterior clinoid processes. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:93.)

It is imperative that a watertight dural closure be performed to prevent the possibility of a cerebrospinal fluid leak. Pericranium or a dural substitute, a tissue sealant, and a fat graft should be used as needed. Any exposed air cells of the temporal bone should be aggressively waxed closed.

17.4.2 Transpetrosal Approach

The transpetrosal approach comprises the retrolabyrinthine, translabyrinthine, and transcochlear approaches, each of which involves various amounts of resection of the lateral petrous and mastoid bones.

Patient positioning is the same as for the middle cranial fossa approach. If the patient's habitus precludes adequate depression of the shoulder, the lateral decubitus–park bench position is used. The horseshoe-shaped skin incision is concave anteriorly and inferiorly. It begins immediately anterior to the tragus and curves around the ear two to three fingerbreadths from the root of the pinna. It ends posteriorly just inferior to the tip of the mastoid process (▶ Fig. 17.7). The scalp flap is reflected anteroinferiorly with fishhooks. Burr holes are made above the root of the zygomatic process and above and below the transverse sinus just proximal to the transverse–sigmoid sinus junction (▶ Fig. 17.8). The location of the sinus is confirmed by locating the asterion, usually using intraoperative image guidance.

First, a high-speed drill with a diamond bit is used to expose the entire sigmoid sinus from the transverse–sigmoid sinus junction to the jugular bulb. Next, the neuro-otologist performs a generous mastoidectomy. From anterosuperiorly to inferoposteriorly, the cortical bone overlying the incus and semicircular canals, the facial nerve, fallopian canal, and endolymphatic sac are exposed (▶ Fig. 17.9). Once these structures are exposed, a presigmoid retrolabyrinthine corridor is established. If the semicircular canals and endolymphatic sac are drilled, a translabyrinthine corridor is established. If the middle ear bones and cochlea are also drilled, with or without translocation of the facial nerve, a transcochlear corridor is established. In the latter case, a small muscle plug is placed to prevent a cerebrospinal fluid leak.

Next, a craniotomy is made using the previously placed burr holes to expose the dura constituting the sinodural angle, which

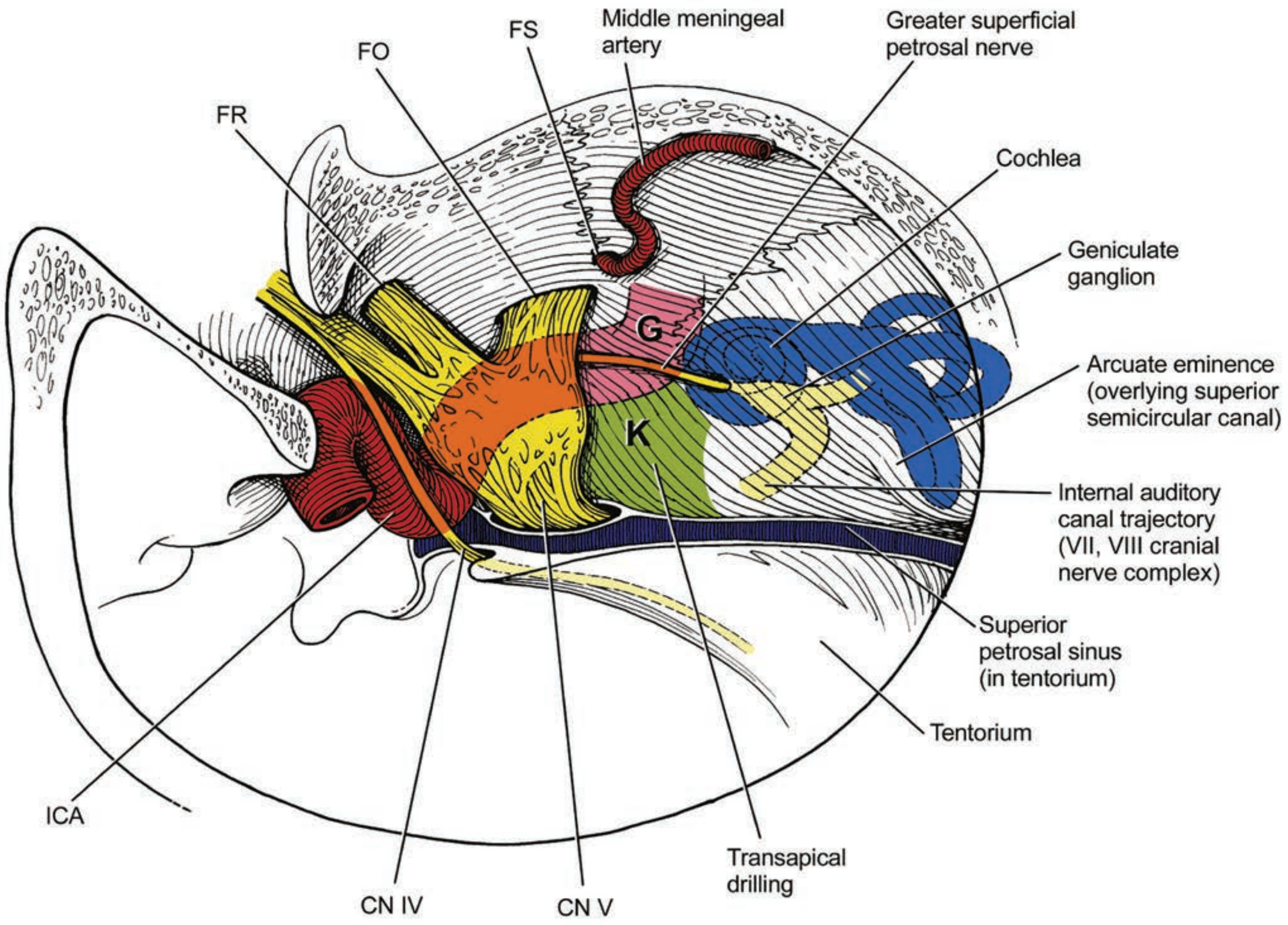
FS
FO
Middle meningeal artery
Greater superficial petrosal nerve
FR
Cochlea
Geniculate ganglion
G
K
Arcuate eminence (overlying superior semicircular canal)
Internal auditory canal trajectory (VII, VIII cranial nerve complex)
Superior petrosal sinus (in tentorium)
Tentorium
ICA
Transapical drilling
CN IV
CN V

Fig. 17.4 Anatomical landmarks that guide drilling for the middle fossa transapical exposure. The anterior margin is the trigeminal nerve (cranial nerve V and the mandibular division exiting through the foramen ovale). The lateral margin is the greater superficial petrosal nerve, the inferior margin is the internal carotid artery (ICA) (under G, Glasscock triangle), and the medial margin is the superior petrosal sinus (in tentorium). Posteriorly, the cochlea should be avoided. FO, foramen ovale; FR, foramen rotundum; FS, foramen spinosum; K, Kawase triangle. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:89.)

is the groove between the dura of the middle cranial fossa above and the dura immediately anterior to the sigmoid sinus at the entry point of the superior petrosal sinus. The latter should be clearly identified. A **Z**-shaped durotomy (▶ Fig. 17.10) begins above the jugular bulb and follows the anterior margin of the sigmoid sinus to just inferior to its junction with the superior petrosal sinus. The durotomy then turns anteriorly toward the semicircular canals, parallel and immediately inferior to the superior petrosal sinus. The superior petrosal sinus is coagulated and divided. Next, the durotomy proceeds laterally to medially across the tentorium to the incisura, preserving the trochlear nerve. The dura is reflected using anterior and superior tack-up sutures (▶ Fig. 17.11). After arachnoid dissection, the superior cerebellar artery, AICA, and midbasilar artery as well as the trochlear and cranial nerves caudal to it should be visible. The vein of Labbé must be preserved to avoid venous infarcts.

Pericranium, local temporalis fascia, or a dural substitute is used along with fat graft and a tissue sealant to obtain a watertight dural closure. The authors often perform a titanium mesh cranioplasty. The incision should be closed in multiple layers with a separate running monofilament suture for the skin.

17.4.3 Retrosigmoid Approach

The retrosigmoid craniotomy is a workhorse approach for a variety of lesions involving the midbasilar artery, including those at the origin of the AICA. It provides a narrow opening along a trajectory that parallels the petrous bone. Positioning is similar to that of the middle fossa and transpetrosal approaches. A curvilinear incision is made approximately two fingerbreadths behind the pinna centered at the transverse–sigmoid junction. It typically extends from the top of the ear to the mastoid tip, but wand navigation is typically helpful in planning this incision (▶ Fig. 17.12).

The retromastoid groove and asterion are identified, and a single burr hole is placed at the transverse–sigmoid junction. A craniotomy or craniectomy is performed with careful attention paid to keeping the dura intact. Bone is removed as laterally as possible to expose the distal part of the transverse sinus, the transverse–sigmoid sinus junction, and the proximal sigmoid sinus (▶ Fig. 17.13). A curved durotomy is made 2 to 3 mm medial to the sinus, and intraosseous tack-up sutures are placed to allow the sinus to be reflected superiorly

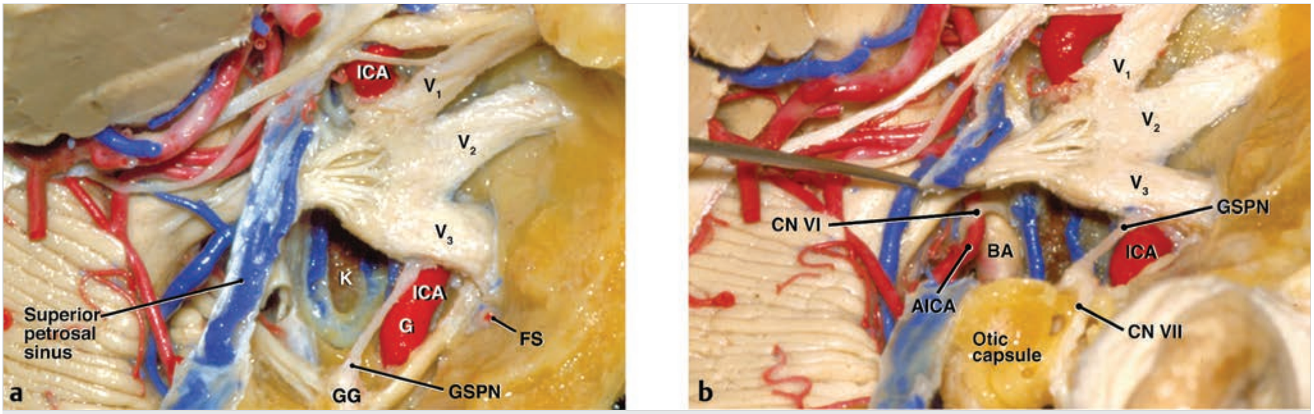

Fig. 17.5 Photographs of a cadaveric dissection show the right middle fossa. **(a)** The exposure shows the Kawase triangle (K) after the petrous apex has been drilled. This area is a corridor into the posterior fossa from the middle fossa. **(b)** Once this door to the posterior fossa has been opened, the midbasilar trunk is exposed. Note the close relationship between the anterior inferior cerebellar artery (AICA) and abducens nerve (cranial nerve [CN] VI). BA, basilar artery; CN VII, facial nerve; FS, foramen spinosum; G, Glasscock triangle; GG, geniculate ganglion; GSPN, greater superficial petrosal nerve; ICA, internal carotid artery; V_1, ophthalmic branch of the trigeminal nerve; V_2, maxillary branch of the trigeminal nerve; and V_3, mandibular branch of the trigeminal nerve. (Reproduced with permission from Gonzalez LF, Amin-Hanjani S, Bambakidis NC, Spetzler RF. Skull base approaches to the basilar artery. Neurosurg Focus 2005;19(2):E3.)

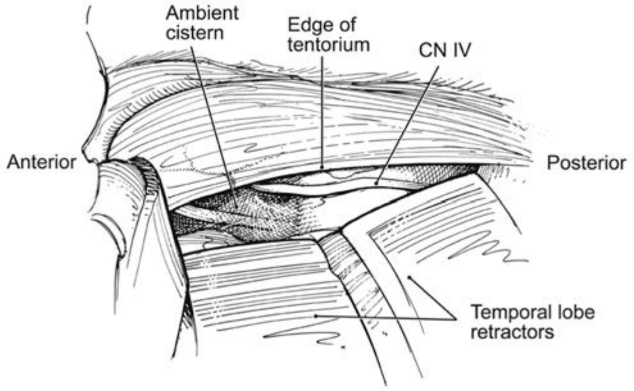

Fig. 17.6 Relationship of the trochlear nerve to the tentorial edge. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:263.)

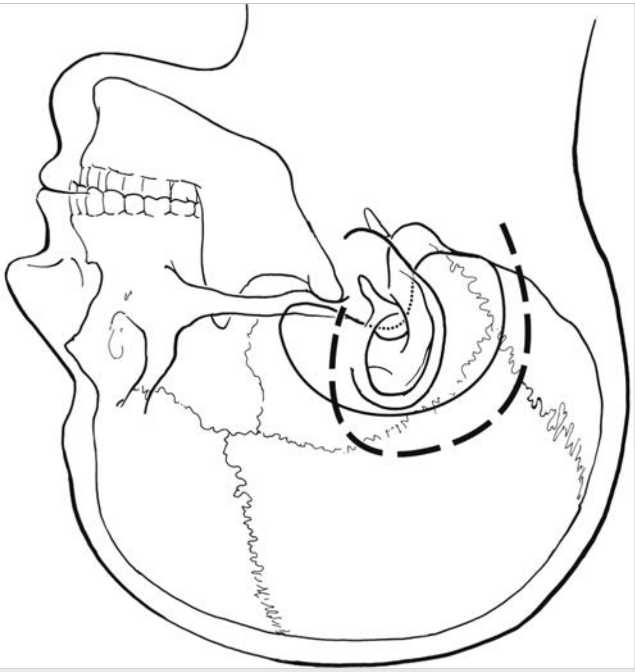

Fig. 17.7 The scalp incision for a transpetrosal approach extends below the zygomatic arch in front of the ear between the frontalis branch of the facial nerve and the tragus of the ear. The posterior rim of the incision extends down to the mastoid tip or beyond. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:96.)

and anteriorly without occluding it. For aneurysms involving the lower two-fifths of the basilar artery, bone resection can be extended inferiorly to the foramen magnum. The cisterna magna is opened and the arachnoid of the cerebellopontine angle is opened sharply downward. A wide arachnoid dissection is performed (▶ Fig. 17.14). At the end of the case, a watertight dural closure should be obtained. Native bone should be restored with snowflake-shaped titanium microplates, or a titanium or other synthetic cranioplasty should be performed.

17.4.4 Far-Lateral Approach

The far-lateral approach provides an oblique and inferior trajectory for exposure of the anterolateral medulla and vertebral artery, from its intradural origin to the confluence, without the need to retract the cerebellum. For aneurysms of the vertebral artery and vertebrobasilar junction, it is not necessary to remove the occipital condyle and expose the extradural vertebral artery. A modified park bench position is used (▶ Fig. 17.15). The head is placed in pins and turned horizontally, then 30 degrees contralaterally toward the dependent shoulder, and declined 20 to 30 degrees

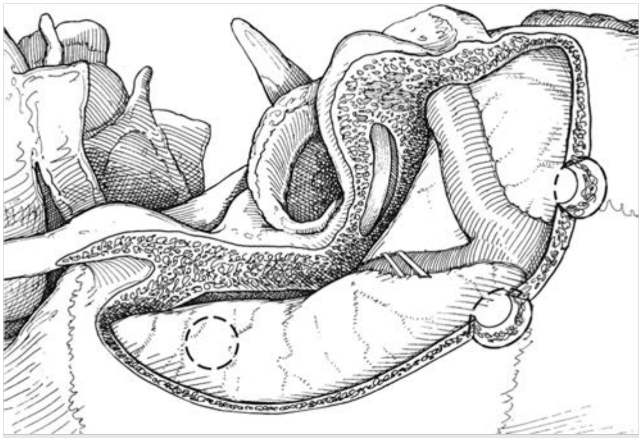

Fig. 17.8 The craniotomy, with burr holes marked, shows preservation of the sigmoid sinus and clips across the superior petrosal sinus. The labyrinthine segment of the facial nerve is exposed after removing the roof of its bony canal. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:88.)

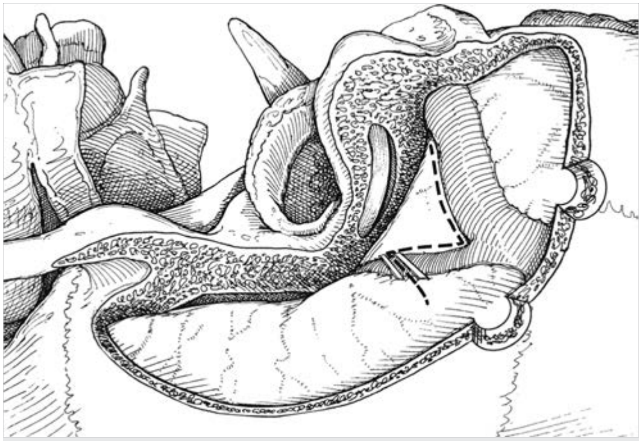

Fig. 17.10 The craniotomy is shown with a line indicating the presigmoid dural incision with preservation of the sigmoid sinus and clips across the cauterized superior petrosal sinus. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:99.)

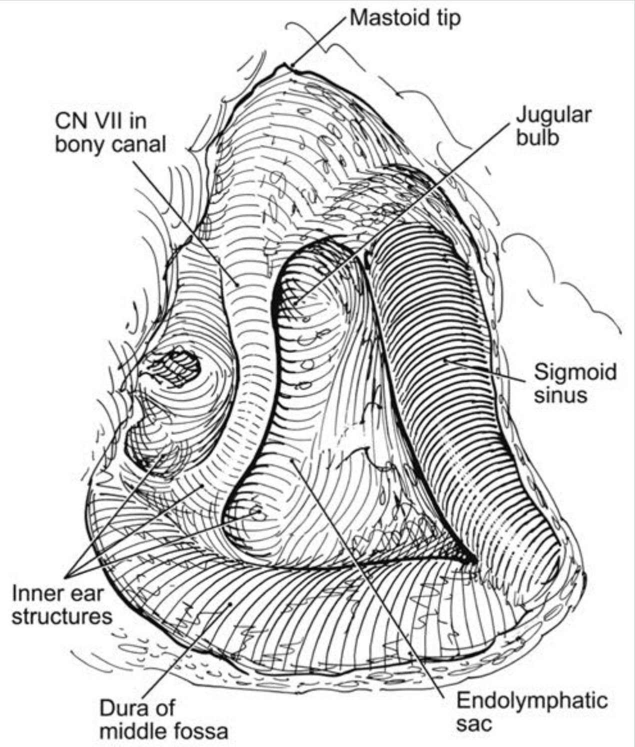

Fig. 17.9 Transpetrosal drilling exposes the sigmoid sinus and the dura of the middle and posterior fossae and outlines the bony canal of the facial nerve. Cortical bone overlying semicircular canals (inner ear structures) is indicated. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:305.)

toward the floor to open the angle between the shoulder and side of the head. The chin is tucked 20 to 30 degrees toward the manubrium.

A paramedian lazy-S incision is made halfway between the mastoid tip and inion. The inflection point is at the level of the foramen magnum. Superiorly, the incision ends two fingerbreadths behind the root of the ear. Inferiorly, it ends in the midline over the spinous process of the third cervical vertebra (▶ Fig. 17.16a). The inion and midline occipital bone keel between the inion and foramen magnum and tubercle of the posterior arch of the atlas and ipsilateral atlas hemilamina and lateral mass are exposed. Injury to the vertebral artery should be avoided by maintaining orientation from the midline and by recognizing the unique darkish yellow fat and rich venous plexus surrounding the vertebral artery in this location. Excessive monopolar cauterization in the region should be avoided to prevent unintended injury to the vertebral artery. Ultrasound can be used to identify the vertebral artery, but is not necessary. To optimize the angle of exposure, the craniotomy should extend from just lateral to the midline to as far laterally as possible and should include removal of the foramen magnum. The venous sinuses do not need to be exposed (▶ Fig. 17.16b). A hemilaminectomy of the atlas is performed. The sigmoid-shaped durotomy starts in the midline below the foramen magnum and curves cephalad and laterally over the inferior cerebellum near the upper lateral margin of the craniotomy (▶ Fig. 17.17). The dura is reflected, the cisterna magna is entered to allow cerebrospinal fluid to drain, and the arachnoid in this region is dissected meticulously and generously. The intradural origin of the vertebral artery is identified and followed cephalad to the confluence (▶ Fig. 17.18). The same closure principles are used as outlined in the approaches already described. Postoperatively, lumbar drainage is recommended for a few days.

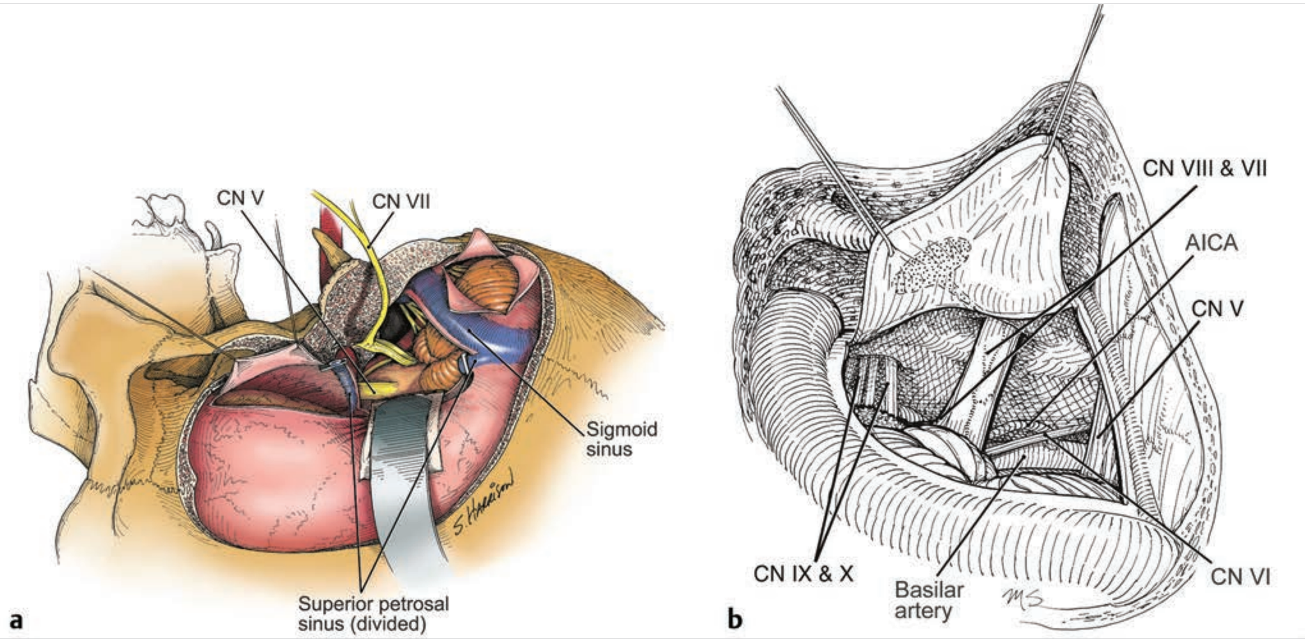

Fig. 17.11 **(a)** The temporal lobe and the incised tentorium, protected by a retractor. The base of the temporal lobe, along with the incised tentorium, is elevated without stretching the great anastomotic vein of Labbé. **(b)** Presigmoid retrolabyrinthine craniectomy with complete skeletonization of the sigmoid sinus, which can be transected if adequate drainage is present contralaterally on the preoperative angiogram or after pressure within the sinus has been determined before and after its temporary occlusion. AICA, anterior inferior cerebellar artery; CN V, trigeminal nerve; CN VI, abducens nerve; CN VII, facial nerve; CN VIII, vestibulocochlear nerve; CN IX, glossopharyngeal nerve; CN X, vagus nerve. (Reproduced with permission from Barrow Neurological Institute, Phoenix, AZ.)

17.5 Postoperative Management Including Possible Complications

Complications associated with the extended middle fossa technique include traction on the GSPN, which can lead to facial paresis. During the temporal durotomy, injuring the vein of Labbé can lead to infarction of the temporal lobe. There is a risk of deafness due to entry into the cochlea during the drilling of the floor of the middle cranial fossa. The internal carotid artery is also at risk during this drilling. Injury to the trochlear nerve during tentorial dural opening can lead to debilitating diplopia.

A lengthy discussion should be had with the anesthetist prior to extubation if lower cranial neuropathy or brain stem functional impairment is anticipated. Early ambulation with physical therapy is recommended. Venous thromboembolic disease is minimized by using graduated compression stockings and subcutaneous pharmacological prophylaxis. To minimize the risk of cerebrospinal fluid leakage, we attempt to obtain watertight dural closure with native dura or a dural substitute. Opened air cells need to be waxed, and a tissue sealant with or without a fat graft is used in some cases. If a cerebrospinal fluid leak occurs, a lumbar drain is recommended and, if persistent, wound re-exploration may be required. The blood pressure is maintained in the normotensive range. For a completely obliterated aneurysm, intraoperative or immediate postoperative angiography is done to confirm this, and follow-up is recommended at 3 and 10 years. If a remnant is detected perioperatively, the first follow-up angiogram is recommended in 1 to 2 years. One can maintain a registry of aneurysm patients to ensure that they are not lost to follow-up.

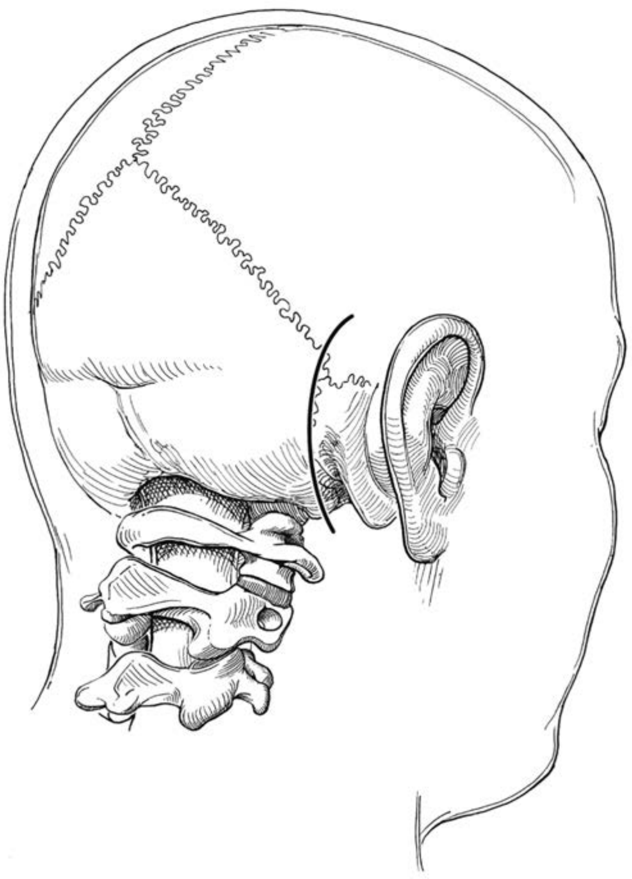

Fig. 17.12 The incision for a retrosigmoid craniotomy. (Reproduced with permission from Barrow Neurological Institute, Phoenix, AZ.)

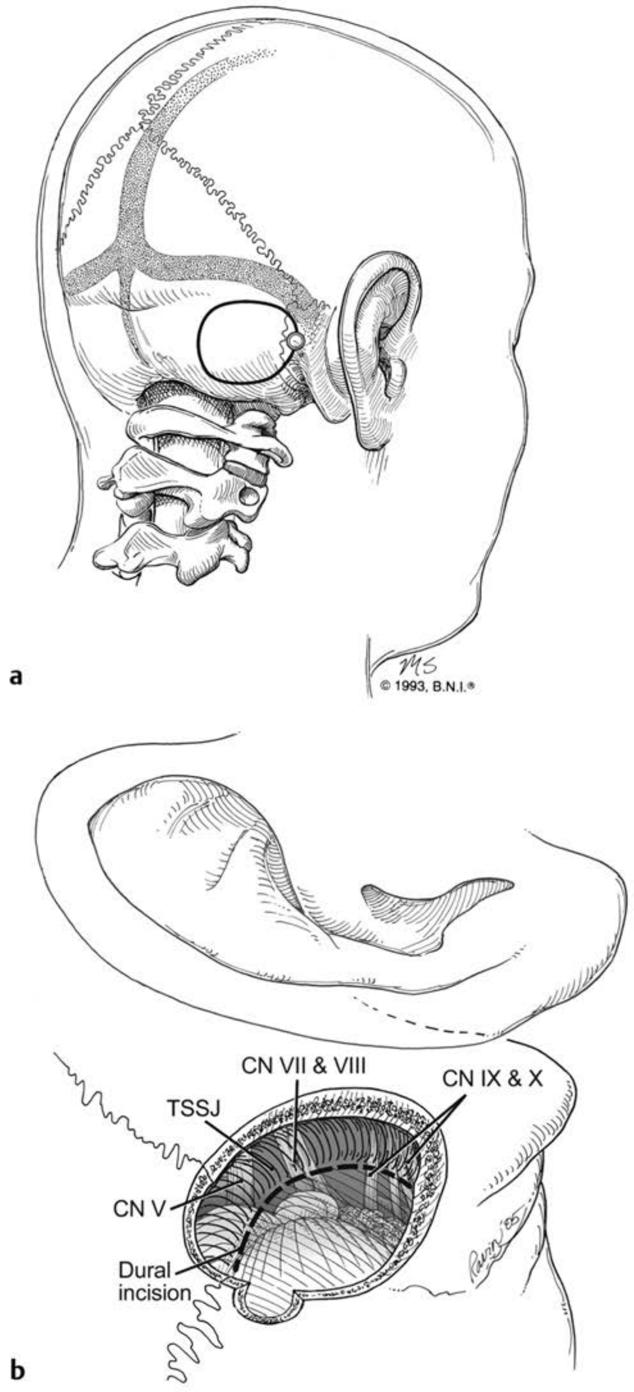

Fig. 17.13 **(a)** The retrosigmoid craniotomy. Care must be taken to expose the sigmoid sinus, which may require resection of mastoid air cells. These should be sealed appropriately. **(b)** Drawing showing a retrosigmoid craniotomy with complete exposure of the sigmoid sinus. Opening the dura mater as close as possible to the sinus allows it to be pulled and the sinus to be retracted to maximize exposure. The basilar artery is located deep at the bottom of the exposure, beneath cranial nerves V to X. TSSJ, transverse–sigmoid sinus junction. (Reproduced with permission from Barrow Neurological Institute, Phoenix, AZ.)

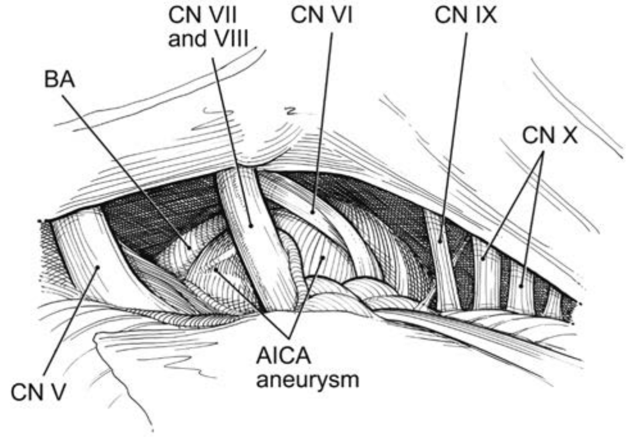

Fig. 17.14 View of an anterior inferior cerebellar artery (AICA) aneurysm through a retrosigmoid craniotomy. The aneurysm is visible below the trigeminal (CN V), facial (CN VII), and vestibulocochlear (CN VIII) nerves. The abducens nerve (CN VI) drapes over the dome of the aneurysm. BA, basilar artery. (Reproduced with permission from Spetzler RF, Daspit CP, Pappas CTE. The combined supra- and infratentorial approach for lesions of the petrous and clival regions: experience with 46 cases. J Neurosurg 1992;76:588–599.)

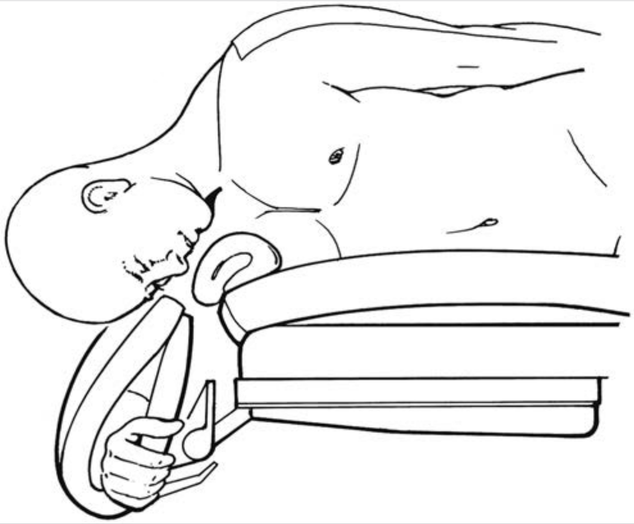

Fig. 17.15 The park bench position. This position allows good exposure of the cerebellopontine angle or midline structures. It is important to pull the ipsilateral shoulder inferiorly and to contralaterally flex the neck (i.e., “decline the head”) ~30 degrees from the horizontal, in addition to rotating the head contralaterally 15 to 30 degrees and tucking the chin maximally. This allows clear access above and behind the ipsilateral shoulder. (Reproduced with permission from Barrow Neurological Institute, Phoenix, AZ.)

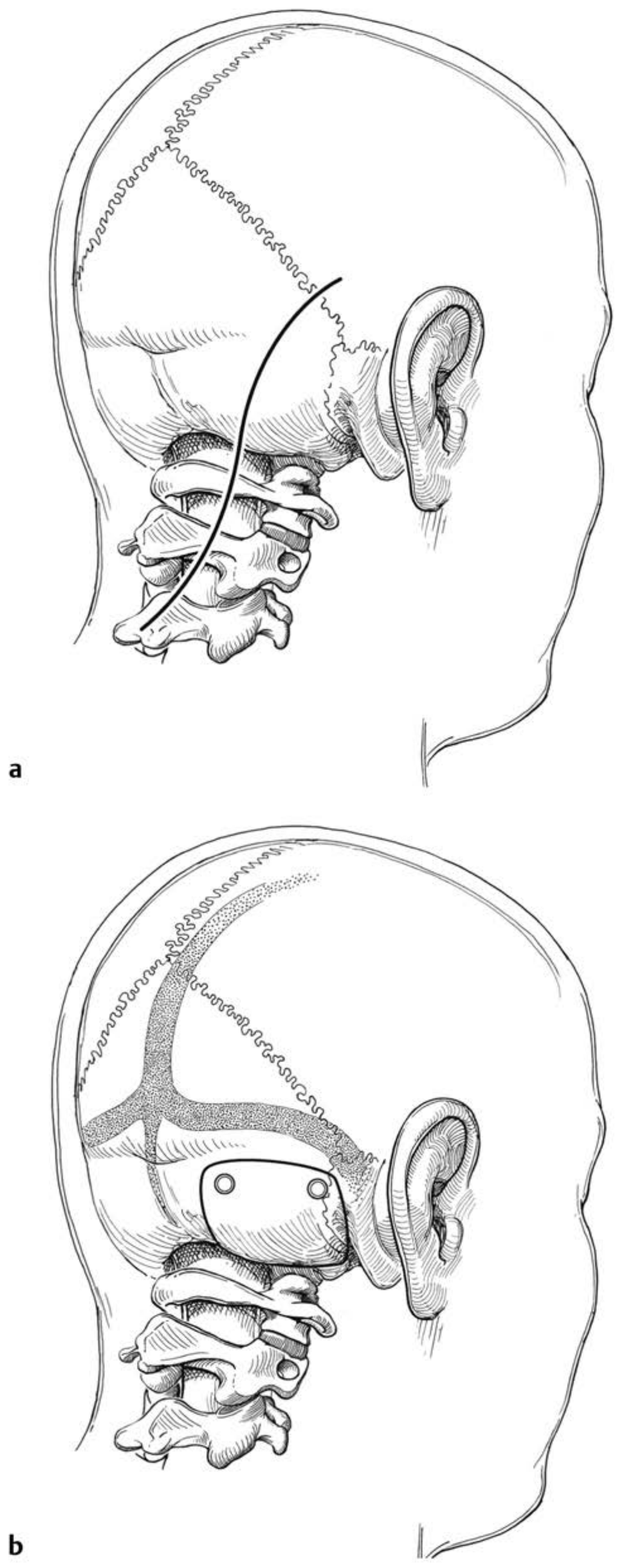

Fig. 17.16 **(a)** The scalp incision for the far-lateral approach. **(b)** The proposed far-lateral craniotomy. (Reproduced with permission from Barrow Neurological Institute, Phoenix, AZ.)

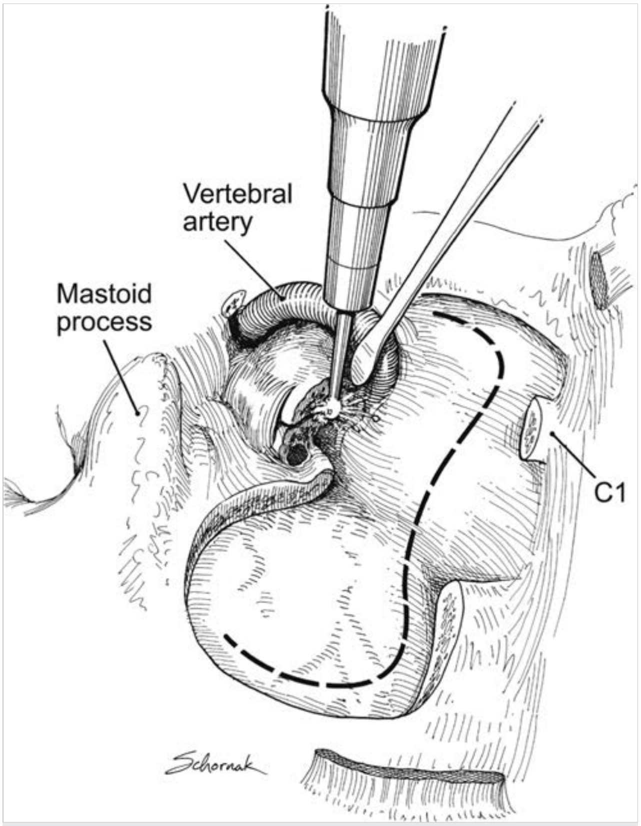

Fig. 17.17 Dural opening for the far-lateral approach. (Reproduced with permission from Baldwin HZ, Miller CG, van Loveren HR, Keller JT, Daspit CP, Spetzler RF. The far lateral/combined supra- and infratentorial approach. A human cadaveric prosection model for routes of access to the petroclival region and ventral brain stem. J Neurosurg 1994;81:60–68.)

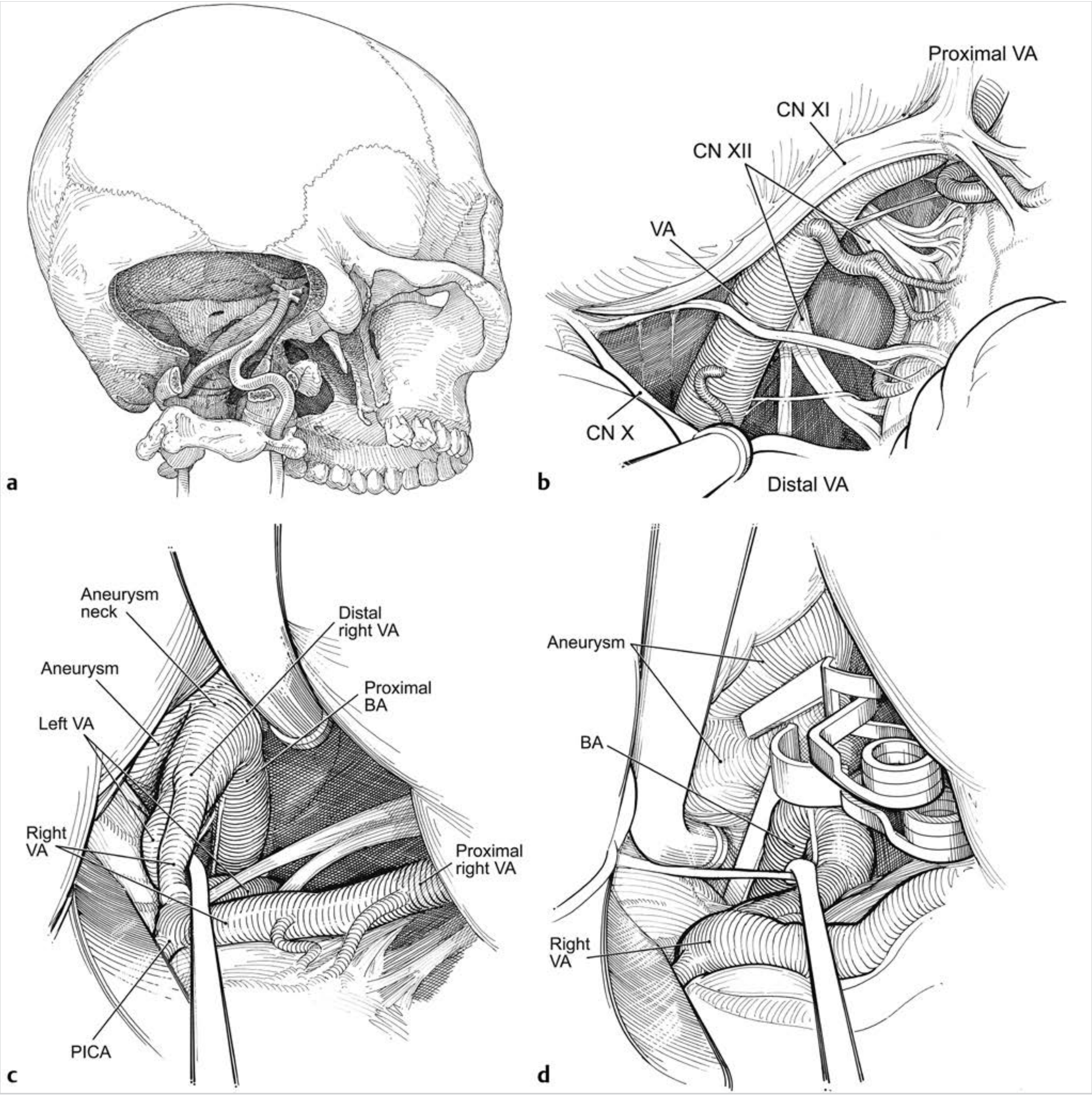
a
Proximal VA
CN XI
CN XII
VA
CN X
Distal VA
b
Aneurysm neck
Distal right VA
Aneurysm
Proximal BA
Left VA
Right VA
Proximal right VA
PICA
c
Aneurysm
BA
Right VA
d

Fig. 17.18 **(a)** Vertebral confluence shown from the view obtained by the far-lateral craniotomy. (Reproduced with permission from Barrow Neurological Institute, Phoenix, AZ). **(b)** Under higher magnification, the vertebral artery (VA) and surrounding cranial nerves are exposed through the far-lateral approach. **(c)** With the patient under hypothermic circulatory arrest, the two vertebral arteries can be seen to form the basilar artery (BA). The neck of the aneurysm and distal basilar artery are visible. **(d)** With a long, 45-degree-angle fenestrated clip, the distal portion of the neck of the aneurysm is occluded parallel to the BA. The residual portion of the neck at the fenestration is obliterated with additional aneurysm clips. PICA, posterior inferior cerebellar artery. (Reproduced with permission from Spetzler RF, Koos WT, Richling B, Lang J. Approaches. In: Spetzler RF, Koos WT, eds. Color Atlas of Microneurosurgery: Microanatomy, Approaches, and Techniques. 2nd ed. Vol. 2: Cerebrovascular Lesions. New York, NY: Thieme; 1997:320–321.)

18 Fusiform, Dolichoectatic, and Dissecting Aneurysms

Christopher M. Owen and Michael T. Lawton

Abstract
Fusiform and dolichoectatic aneurysms are uncommon compared with saccular aneurysms, and they are also different in their clinical presentation and treatment options. Dissecting aneurysms of the intracranial circulation are extremely rare apart from vertebral artery dissections, and their treatment is thus not well defined. These aneurysms can range from small fusiform dilations of a single vessel to giant dolichoectatic aneurysms incorporating the origins of multiple arteries and filled with thrombus. Successful elimination of these aneurysms often requires complex neurovascular techniques such as trapping with bypass of distal vessels, proximal occlusion, resection with reanastomosis, transposition, or thrombectomy and clip reconstruction.

Keywords: aneurysm, fusiform aneurysm, dolichoectatic aneurysm, dissecting aneurysm, vascular bypass

18.1 Introduction

Fusiform and dolichoectatic aneurysms are uncommon compared with saccular aneurysms, and they are also different in their clinical presentation and treatment options. These aneurysms can range from small fusiform dilations of a single vessel to giant dolichoectatic aneurysms incorporating the origins of multiple arteries and filled with thrombus. They lack a definable neck that can be clipped, and often the vessel of origin is circumferentially involved in the aneurysmal dilation or is encompassed in a large, thrombosed mass with distal branches emerging from the aneurysm wall. Successful elimination of the aneurysm often requires trapping with bypass of distal vessels, proximal occlusion, resection with reanastomosis, transposition, or thrombectomy and clip reconstruction.[1]

Dissecting aneurysms of the intracranial circulation are extremely rare apart from vertebral artery dissections, and their treatment is thus not well defined. Some fusiform aneurysms likely have an underlying dissecting etiology.

18.2 Patient Selection

18.2.1 Fusiform and Dolichoectatic Aneurysms

Fusiform or dolichoectatic aneurysms cause symptoms in three ways: compression, ischemia, or rupture. Compared with saccular aneurysms, dolichoectatic aneurysms are more likely to cause compression or ischemia and may be less likely to rupture.

If a patient presents with symptoms suggestive of hemorrhage, then computed tomography (CT) is diagnostic. If the CT is negative, then a lumbar puncture is warranted. For patients who present with new neurological deficits, CT may show ischemia or edema, but magnetic resonance imaging is usually required. CT and CT angiography can also be used to better define an aneurysm's relationship to the skull base prior to surgery.

Catheter angiography in multiple projections, preferably with 3D computer reconstruction, remains the gold standard for diagnosis and surgical planning. Preoperative angiographic studies determine: (1) the artery of origin; (2) the aneurysm's size, shape, and relationship to afferent and efferent arteries; (3) the presence and location of vasospasm or hypoperfusion; (4) the displacement of adjacent vessels suggesting mass effect from hematoma or partial thrombosis of an aneurysmal sac whose dimensions are larger than those seen on angiography; (5) the degree of collateral supply to territory distal to the aneurysm, and (6) the presence of other aneurysms or vascular abnormalities.. We also use magnetic resonance imaging and contrast-enhanced magnetic resonance angiography in the evaluation of giant fusiform and dolichoectatic aneurysms to evaluate their compressive effect on adjacent structures and to visualize organized thrombus within the aneurysm that is not visualized angiographically.

The goals of surgery are to eliminate the risk of hemorrhage, reduce mass effect and compression of adjacent brain and cranial nerves, and preserve normal arterial circulation distal to the aneurysm.[2] The indications for surgery have to balance the risks of treatment with the natural history of these aneurysms. The decision to operate considers the patient's age, medical comorbidities, presenting symptoms, and the size, location, and configuration of the aneurysm. Nonoperative treatment options include antiplatelet therapy for patients with ischemic symptoms, anticonvulsants for patients with neurovascular compression syndromes, or serial imaging observation for asymptomatic or minimally symptomatic lesions. The natural history of fusiform aneurysms is notoriously poor, with 5-year mortality rates > 20% for posterior circulation lesions, and failure of conservative management justifies intervention for these aneurysms.

18.2.2 Dissecting Aneurysms

Anterior circulation and basilar artery dissecting aneurysms classically present with acute focal neurological deficits, whereas intradural vertebral artery dissections often cause subarachnoid hemorrhage (SAH). Blister aneurysms of the dorsal carotid artery are dissecting pseudoaneurysms of the supraclinoid segment, often present with SAH, and have high rates of early rerupture and intraoperative rupture.[3] Angiography is the principal method for detecting these lesions but CT, CT angiography, and magnetic resonance imaging are typically obtained also. The goal of surgery for ruptured lesions is to prevent rebleeding while preserving the circulation distal to the aneurysm. The dissection should generally be trapped. Trapping may be complicated by the presence of eloquent branches along the dissected arterial segment. If necessary, excision with reanastomosis or extracranial–intracranial bypass can be done to preserve circulation.

Pseudoaneurysms due to intracranial dissection have a high rerupture rate, and timely treatment is indicated unless recovery from the initial hemorrhage seems unlikely. Unruptured dissections presenting with ischemia may be treated medically and surgery reserved for symptoms due to mass effect, although management of these has to be individualized.

18.3 Preoperative Preparation

Electroencephalographic and evoked potential monitoring are used in all cases. Barbiturate or propofol burst suppression is used for cerebral protection during temporary clipping. Intravenous adenosine or rapid ventricular pacing can provide a sufficient window of circulatory arrest without the morbidity of hypothermia and cardiopulmonary bypass.

Whenever a parent artery is to be surgically occluded, revascularization of the involved vascular territory must be considered to prevent ischemic complications. Angiographic assessment of collateral blood flow is essential but a balloon test occlusion helps identify patients who are unlikely to tolerate permanent arterial sacrifice. Patients with poor tolerance to occlusion typically require a high-flow bypass such as a saphenous vein or radial artery bypass (▶ Fig. 18.1). An ultrasonographic Allen's test is performed in patients considered for high-flow bypass in whom radial artery harvest may be required. Patients with marginal tolerance may need only a low-flow bypass. In the setting of SAH, the threshold for revascularization is lower, as even a successful test occlusion does not ensure that perfusion will be adequate if vasospasm develops.

18.4 Operative Procedure

The size, shape, and location of the aneurysm determine the surgical approach. A pterional craniotomy with extensive drilling of the sphenoid wing provides access to most anterior circulation aneurysms. Occasionally, an orbitozygomatic osteotomy is needed to increase exposure. The superficial temporal artery (STA) must be preserved during the approach. Dissecting aneurysms have a high intraoperative rupture rate, and adequate proximal control is assured by removal of the anterior clinoid process or exposure of the cervical internal carotid artery (ICA). Aneurysms on the second and third segments of the anterior cerebral artery require an interhemispheric approach. Aneurysms of the vertebral artery or vertebrobasilar junction are approached through a far-lateral (transcondylar) craniotomy with laminectomy of the atlas. Midbasilar aneurysms are approached through a transpetrosal route that removes portions of the petrous bone (retrolabyrinthine, translabyrinthine, or transcochlear). With smaller midbasilar aneurysms, an extended retrosigmoid approach may be sufficient and can avoid the complications associated with a transpetrosal approach. When greater exposure is needed, the transpetrosal approaches can be combined with a subtemporal craniotomy and division of the tentorium.

18.4.1 Clip Reconstruction of the Parent Artery

By definition, fusiform and dolichoectatic aneurysms lack a neck, making clip reconstruction more difficult than for simple saccular aneurysms. Typically, multiple clips and fenestrated clips are needed, and the aneurysm has to be temporarily trapped to accomplish clip reconstruction. These aneurysms are also frequently filled with thrombus. To clip the aneurysm, the mass effect must first be reduced by thrombectomy (▶ Fig. 18.2). Thrombectomy requires opening the aneurysm

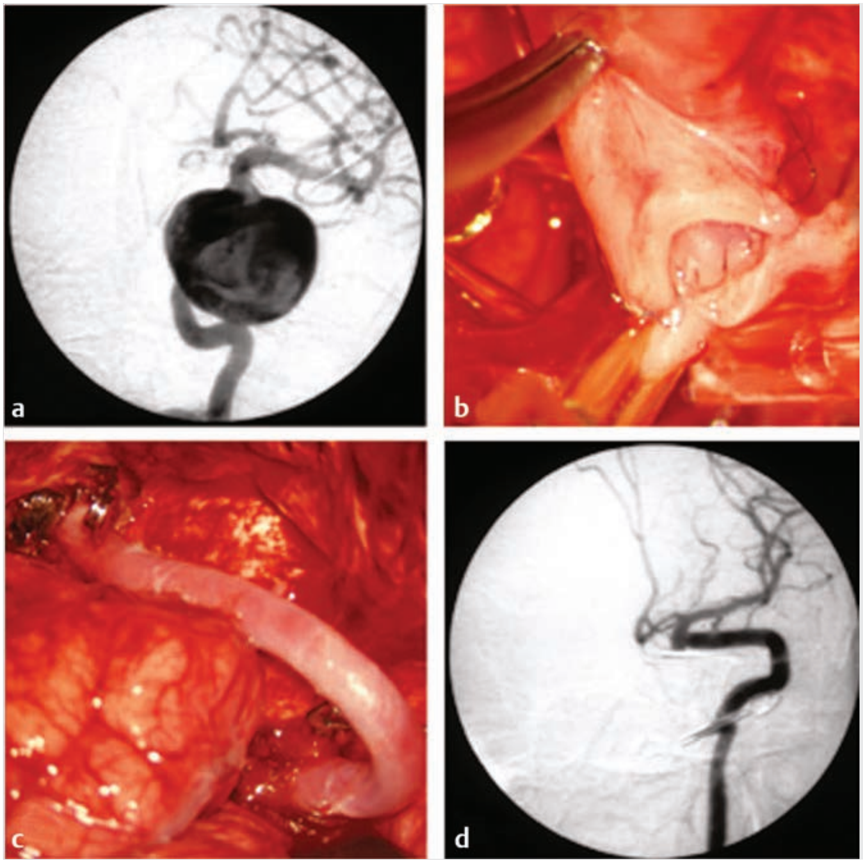

Fig. 18.1 A 15-year-old boy presented with double vision. A left cavernous internal carotid artery aneurysm was seen on **(a)** left internal carotid artery (ICA) angiography (anteroposterior view). A balloon test occlusion of his left ICA with hypotensive challenge and a single photon emission computed tomography cerebral blood flow study demonstrated asymptomatic diminished perfusion to his left hemisphere. The aneurysm was therefore treated with surgical trapping and petrous-to-supraclinoid ICA bypass. **(b)** A proximal end-to-side anastomosis was performed using a short saphenous vein graft, and a distal end-to-side anastomosis to the supraclinoid ICA was performed. **(c)** The completed bypass enabled the cavernous aneurysm to be trapped, and **(d)** the postoperative angiogram shows good reconstitution of blood flow to the left hemisphere.

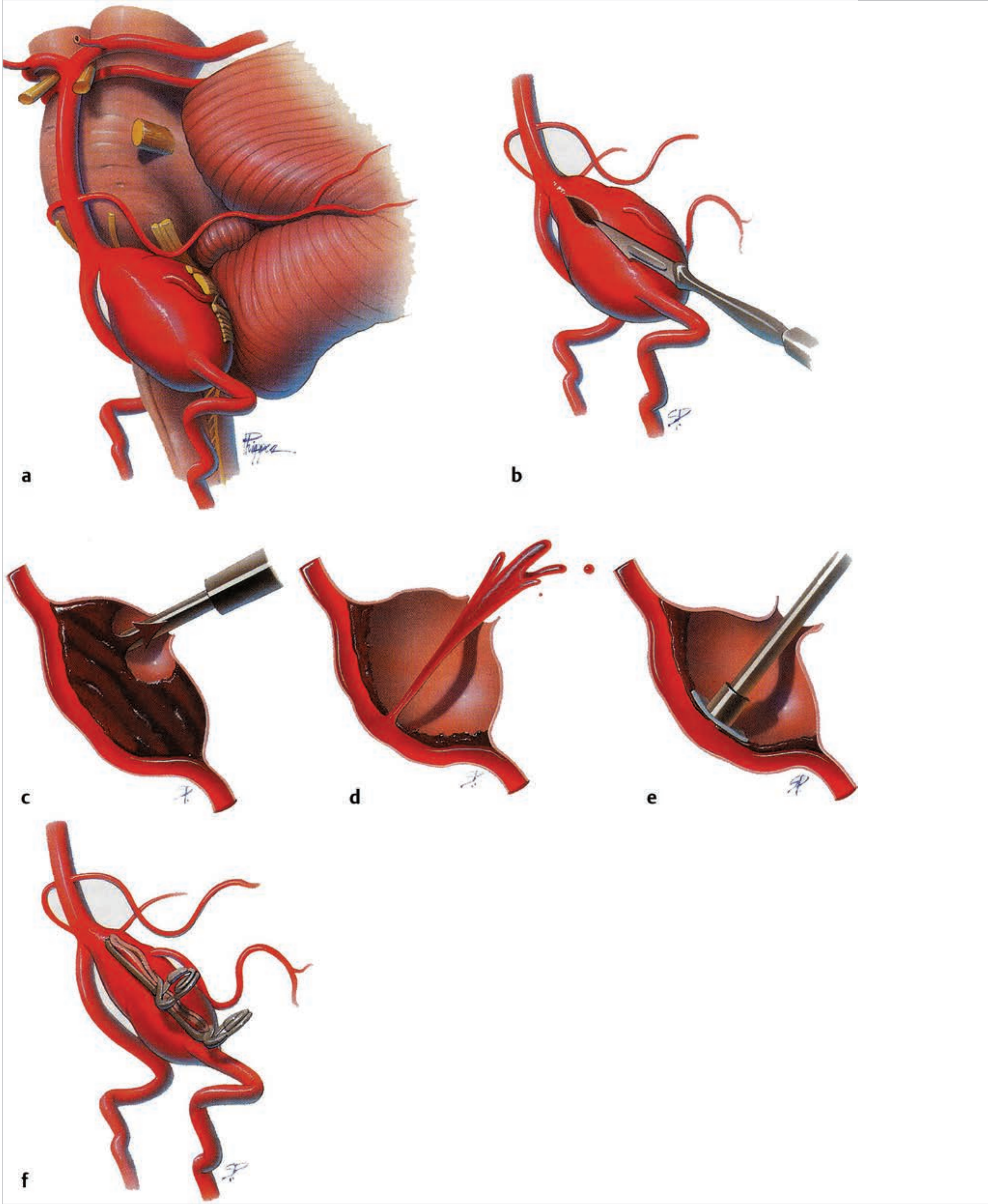

Fig. 18.2 **(a)** Dolichoectatic aneurysms involving the intradural vertebral artery typically produce symptoms from compression of the brainstem and cranial nerves. **(b)** This compressive mass is eliminated by opening the aneurysm and **(c)** removing thrombus with an ultrasonic aspirator. **(d)** The thrombectomy proceeds until the lumen of the aneurysm is encountered. **(e)** Bleeding is controlled with oxidized cellulose or a comparable substance and **(f)** the walls of the aneurysm are brought together with clips to reconstruct the parent artery. Note that the thrombectomy eliminates mass effect and generates the redundant vessel wall needed to reconstruct the artery. (Reproduced with permission from the Barrow Neurological Institute, Phoenix, AZ.)

under local or global circulatory arrest. Thrombus is removed piecemeal with cup forceps or an ultrasonic aspirator and proceeds until the thrombus has been adequately debulked or until the lumen of the aneurysm is encountered, at which point bleeding is controlled with application of a hemostatic agent (oxidized cellulose) and gentle pressure. It can be difficult to determine if the reconstructed channel through the aneurysm has a sufficient internal caliber to supply the distal branches, so clips are applied to leave a generous lumen, and intraoperative Doppler velocity measurements and angiography are useful.

In the case of dissecting blister aneurysms of the dorsal carotid wall, the dissection usually involves only a portion of the artery circumference and direct clip reconstruction is the preferred treatment. The segment is temporarily trapped, which typically requires temporary clip placement proximally on the cervical or clinoidal ICA, and distally at the ICA terminus. In select cases, temporary clipping of the ophthalmic and/or posterior communicating artery may also be needed to reduce turgor within the segment sufficiently to allow permanent clip reconstruction. Clip blades are applied parallel to the parent artery and must oppose the normal intima, often narrowing the parent lumen by 15 to 30% as the diseased section of wall is excluded (▶ Fig. 18.3). If the carotid is circumferentially diseased, clip reconstruction may not be possible, and contingency maneuvers such as clip reinforced wrapping, or trapping along with high-flow bypass (typically cervical carotid–middle cerebral artery [MCA]) must be prepared for in advance.

18.4.2 Aneurysm/Parent Artery Occlusion

Parent artery occlusion is the simplest method to treat dissecting, fusiform, or dolichoectatic aneurysms (▶ Fig. 18.4). It may involve occlusion of inflow (hunterian ligation), of outflow, or both (trapping). Trapping is ideal because this minimizes the risk of rupture or growth and is theoretically safe if there are no branches from the trapped segment and if collateral blood flow to distal territories is adequate. The first requirement cannot always be met, particularly along the supraclinoid ICA, the sphenoidal segment of the MCA, and the basilar artery. In such cases, it is advisable to use only proximal occlusion. The second requirement (adequate collateral blood flow) may need to be assessed by balloon test occlusion. Distal parent artery occlusion only should not be done on ruptured aneurysms (▶ Fig. 18.4). It is reserved for large and giant dolichoectatic aneurysms where exposure of the proximal artery is difficult (e.g., basilar trunk aneurysms) and the aneurysm is thick walled and/or thrombus filled, with low risk of rupture. Parent artery occlusions can sometimes be performed endovascularly.

18.4.3 Revascularization of the Anterior Circulation

A wide variety of bypasses are available; the choice depends on the location of the aneurysm and the flow requirements of the distal territory (▶ Fig. 18.5).[4] Aneurysms of the infraclinoid ICA can be trapped and bypassed by several methods (▶ Fig. 18.5).

Revascularization after occlusion of the supraclinoid ICA can be achieved with an STA–MCA bypass. Patients with poor tolerance of balloon test occlusion will likely require a high-flow saphenous bypass from the cervical ICA to the MCA. An STA–MCA bypass is also used to revascularize the distal MCA territory after the trapping of MCA aneurysms. A “double-barrel” STA–MCA bypass, which connects both frontal and temporal branches of the STA to different branches of the MCA, can be used to create higher flow to this territory or can be used to revascularize both trunks of the MCA bifurcation.[5] If the STA is damaged during the craniotomy, or is too narrow to provide sufficient flow for bypass, then an in situ bypass can anastomose the anterior temporal artery side-to-side to an MCA branch distal to the aneurysm. Similarly, an uninvolved MCA branch can serve as a donor vessel to an adjacent MCA branch that has been occluded proximally, using either an end-to-side implantation or a side-to-side anastomosis. The double reimplantation technique is another useful technique for completely reconstructing the MCA bifurcation (▶ Fig. 18.6). With this technique, two efferent arteries are reimplanted onto a bypass graft connected to a proximal donor artery.

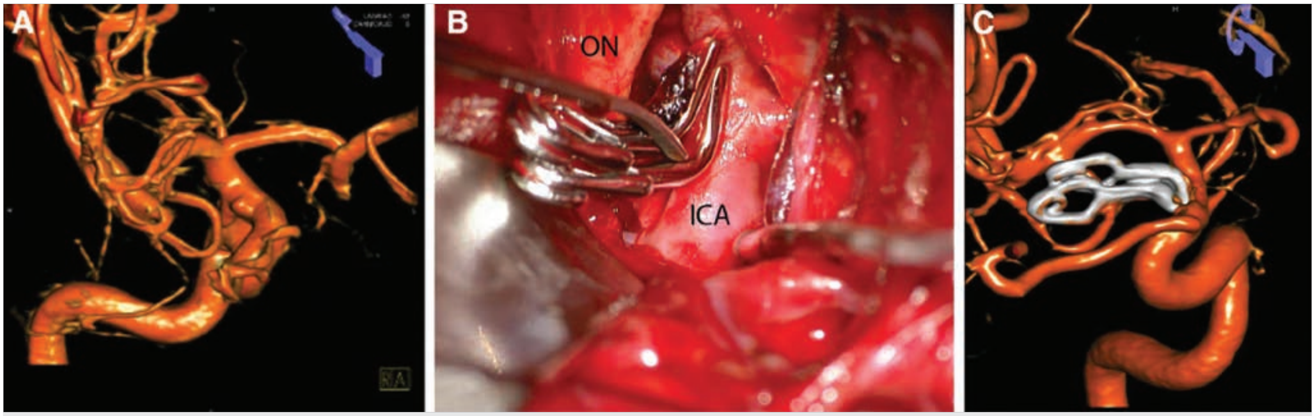

Fig. 18.3 **(a)** Preoperative three-dimensional angiographic reconstruction (right ICA) demonstrated a dorsal ICA blister aneurysm. **(b)** The ICA blister aneurysm was clipped with two stacked right-angled clips with the blades paralleling the axis of the ICA. **(c)** Postoperative three-dimensional angiographic reconstruction showed the stacked angled clips and aneurysm occlusion. ICA, internal carotid artery; ON, optic nerve. (Reproduced with permission from Owen et al.[3])

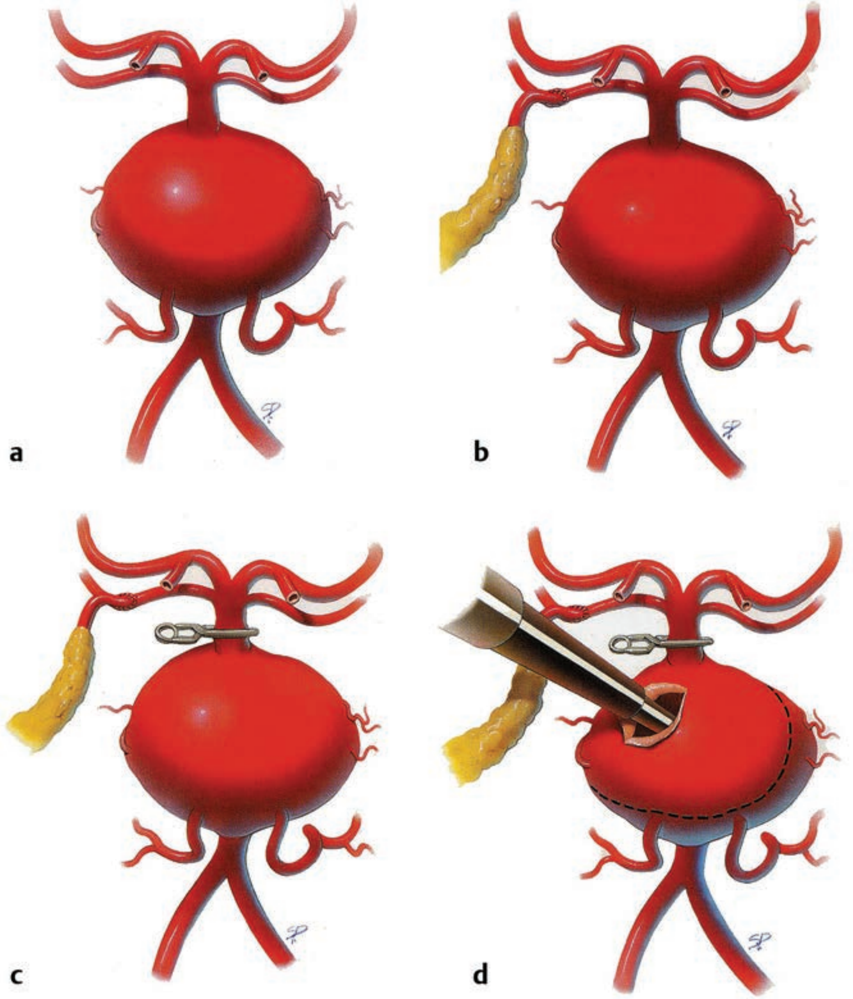

Fig. 18.4 **(a)** Certain dolichoectatic aneurysms, such as midbasilar aneurysms, are difficult to access surgically and their treatment is limited to parent vessel occlusion. **(b)** In an initial operation, blood flow to the distal basilar artery territory is augmented with a superficial temporal artery-to-superior cerebellar artery bypass. **(c)** The distal basilar artery is then occluded with a clip during a second operation. **(d)** Reduction of flow through the aneurysm promotes thrombosis of its lumen and enables a thrombectomy to be performed during a third operation. The dashed line designates the extent of the thrombectomy. Treatment relieves mass effect on the brainstem and preserves distal blood flow. (Reproduced with permission from the Barrow Neurological Institute, Phoenix, AZ.)

If an aneurysm involves one of the anterior cerebral arteries distal to the anterior communicating artery, then a side-to-side anastomosis between the involved anterior cerebral and its contralateral artery can preserve distal flow through both arteries.

18.4.4 Revascularization of the Posterior Circulation

Several bypasses are available to revascularize the posterior circulation (▶ Fig. 18.7).[2] The upper basilar artery can be revascularized using the STA as a donor artery and either the superior cerebellar or posterior cerebral artery. The superior cerebellar is preferred because temporary occlusion is better tolerated. High-flow saphenous vein grafts and intermediate-flow radial artery grafts can also revascularize the basilar apex when the STA is insufficient, using either the cervical carotid artery or the MCA as the donor. If direct access to a basilar artery aneurysm is needed during the same operation as the bypass, then a combined supra- and infratentorial approach is used, and a bypass from the vertebral artery to the superior cerebellar or posterior cerebral artery is performed. Once the bypass is completed, this approach provides direct access to the aneurysm for trapping or proximal occlusion, which is not usually available when traditional approaches to the basilar apex, such as the orbitozygomatic-pterional approach, are used.

The midbasilar trunk and anteroinferior cerebellar artery aneurysms are revascularized with the occipital artery. Rarely will a high-flow bypass be needed here. The V3 segment of the vertebral artery is accessible along the sulcus arteriosus and may be conveniently exposed here during the far-lateral and retrosigmoid approaches, making it a possible donor artery for use with an interposition graft via an end to side anastomosis.

The posterior inferior cerebellar artery (PICA) frequently originates from the base of aneurysms along the V4 segment, and its perforators to the anterior and lateral medulla make preservation of its flow a priority. When trapping a fusiform vertebral artery aneurysm that involves the PICA origin, the PICA can be revascularized with the occipital artery or the contralateral PICA in a side-to-side PICA–PICA bypass, or by reimplantation onto the proximal vertebral artery. Reconstitution of flow in the distal PICA enables this artery to be occluded as it exits the aneurysm, after which retrograde filling from the bypass perfuses the medullary perforators. In cases of fusiform vertebral aneurysms in which the contralateral vertebral is atretic or absent, the aneurysm may be excised and the basilar trunk revascularized with an interposition graft from V3 to the distal V4.

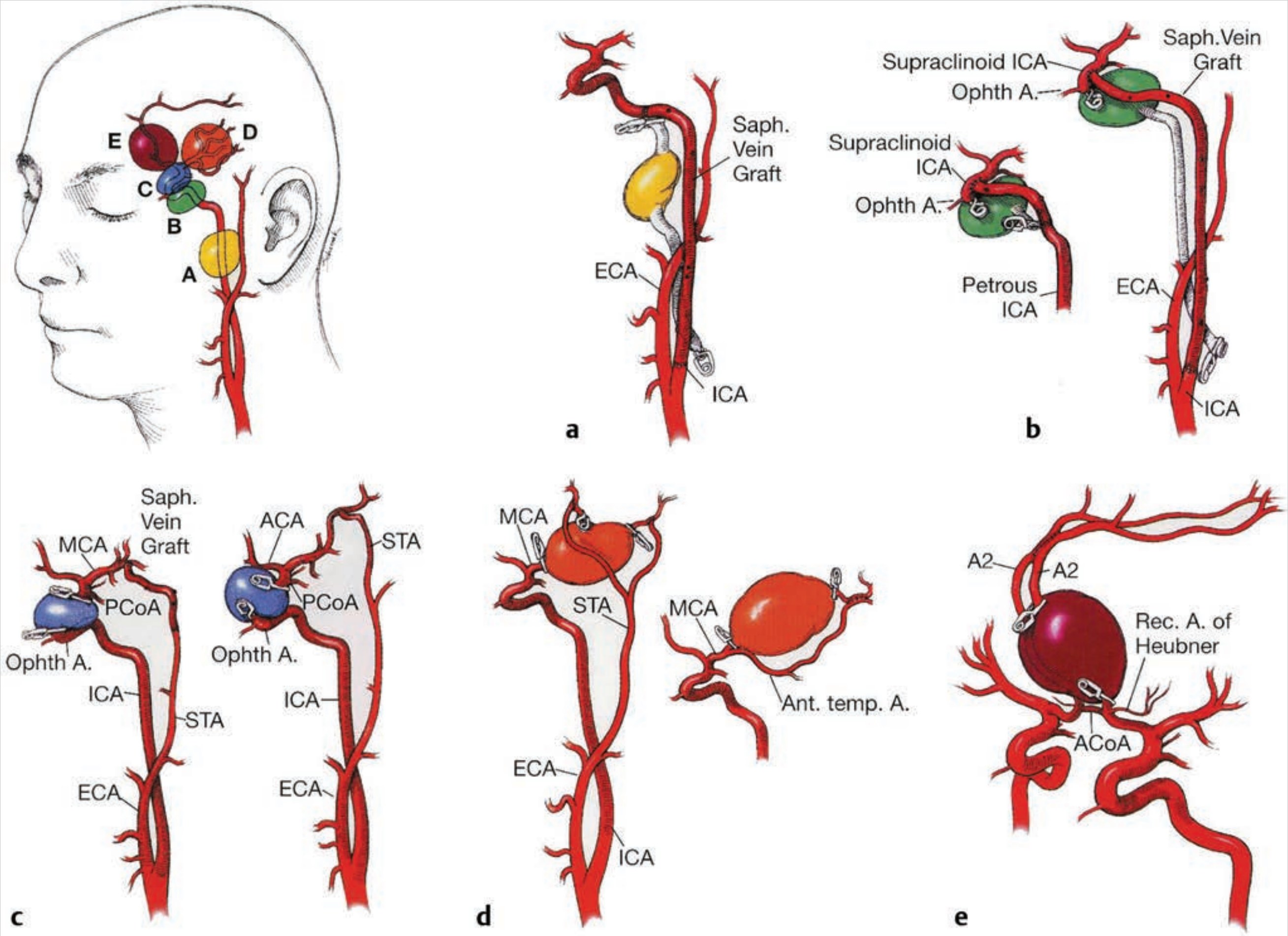

Fig. 18.5 Surgical approaches to revascularization of the anterior circulation. **(a)** Internal carotid artery (ICA) aneurysm at the skull base is trapped and revascularized with a cervical-to-petrous carotid artery saphenous vein bypass. **(b)** Cavernous ICA aneurysm is trapped and revascularized with a petrous-to-supraclinoid carotid artery saphenous vein bypass or a cervical-to-supraclinoid carotid artery bypass. **(c)** The supraclinoid ICA is trapped around an aneurysm on this segment and revascularized with a superficial temporal artery (STA)–middle cerebral artery (MCA) bypass or an STA–MCA saphenous vein interposition vein bypass. **(d)** MCA aneurysm is trapped and revascularized with a “double-barrel” STA–MCA bypass or an anterior temporal artery-to-MCA in situ bypass. **(e)** Anterior cerebral artery aneurysm is trapped and revascularized with an in situ bypass between the second segments of the anterior cerebral arteries. ECA, external carotid artery; Saph, saphenous; Ophth A., ophthalmic artery; PCoA, posterior communicating artery; Ant. temp. A., anterior temporal artery; Rec. A., recurrent artery of Heubner; ACoA, anterior communicating artery. (Reproduced with permission from the Barrow Neurological Institute, Phoenix, AZ.)

18.4.5 Aneurysm Resection and Reanastomosis

Some fusiform and dolichoectatic aneurysms can be resected and the ends of the involved artery proximal and distal to the aneurysm reanastomosed primarily (▶ Fig. 18.8). This technique is only useful for certain aneurysms, usually on the MCA, anterior cerebral, or PICA. When the ends of the arteries cannot be reapproximated without undue tension, a short segment of radial artery or STA provides a convenient interposition graft. When multiple efferent arteries are present, this technique cannot be used.

18.4.6 Mobilization and Transposition

Some fusiform aneurysms may cause compressive symptoms similar to giant aneurysms, especially when they involve arteries in the posterior circulation. An appropriate treatment may be macrovascular decompression that mobilizes the aneurysm and transposes it away from the brainstem and cranial nerves. This can be accomplished by fashioning a sling of muslin or suture around the aneurysm and securing it to the clival dura so that the vessel is lifted off neural structures. A fenestrated clip can also be used as a sling.

18.4.7 Wrapping

Wrapping fusiform aneurysms with muslin or cotton can reinforce the wall and promote an inflammatory reaction that produces tough scar tissue. This theoretically reduces the risk of rupture, although there are few data to support this notion or to demonstrate any reduction in the rate of aneurysm growth. When wrapping blister aneurysms, a sling of muslin

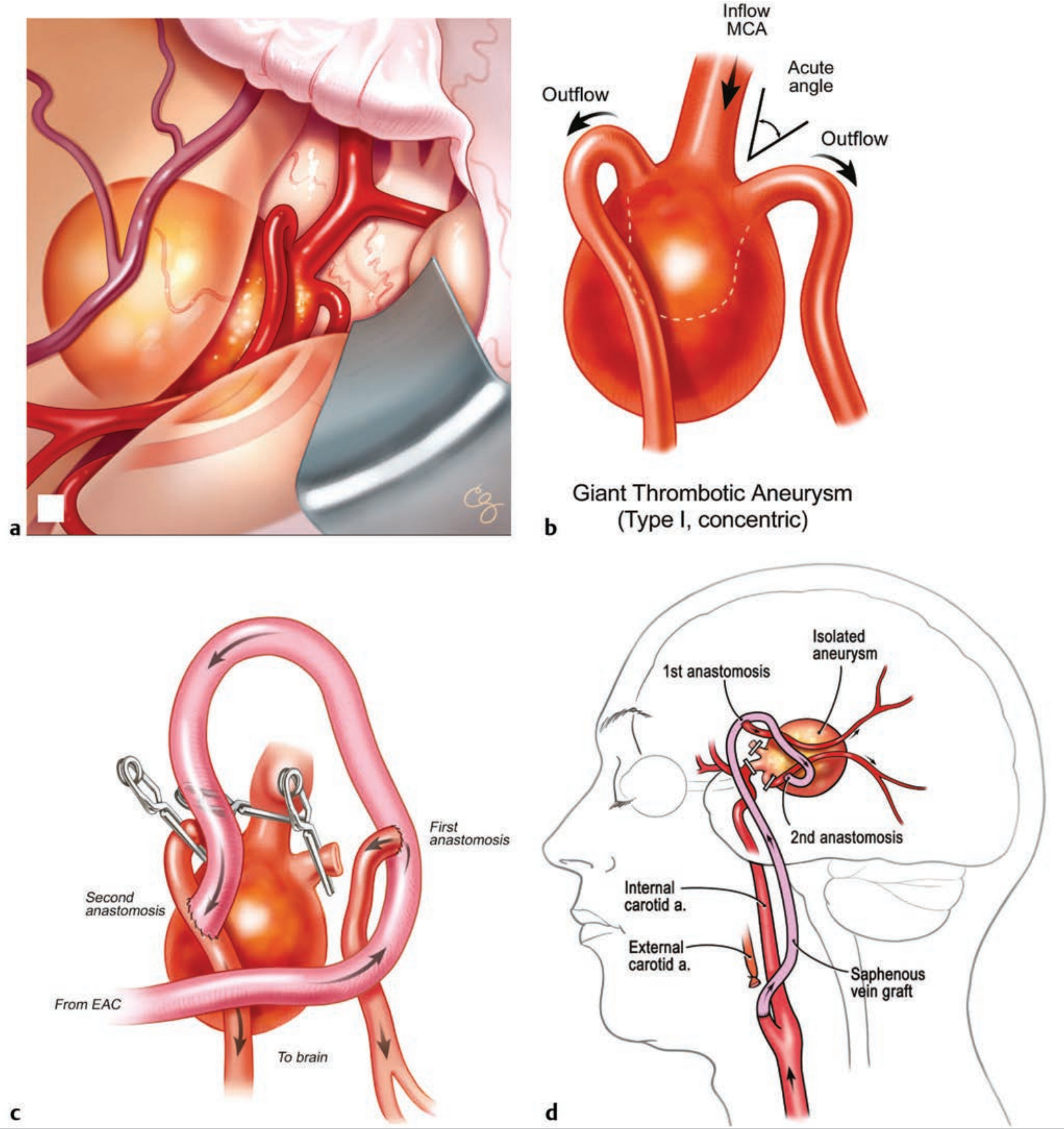
a
Inflow
MCA
Acute
angle
Outflow
Outflow
b
Giant Thrombotic Aneurysm
(Type I, concentric)
First
anastomosis
Second
anastomosis
From EAC
To brain
c
Isolated
aneurysm
1st anastomosis
2nd anastomosis
Internal
carotid a.
External
carotid a.
Saphenous
vein graft
d

Fig. 18.6 Double reimplantation surgical technique. **(a)** Surgical exposure of the giant, thrombosed middle cerebral artery (MCA) aneurysm through a standard left pterional craniotomy, as seen from the neurosurgeon's perspective intraoperatively. **(b)** Relationship between the afferent sphenoidal MCA segment and efferent trunks originating from the base of the aneurysm at acute angles. The lumen of the aneurysm is depicted with a dashed line, with the remainder of the aneurysm filled with thrombus. **(c)** After first connecting the saphenous vein graft to the cervical external carotid artery with an end-to-end anastomosis, the first MCA branch is isolated between aneurysm clips, transected, and reimplanted on the bypass graft with an end-to-side anastomosis. The temporary clip on bypass graft is repositioned beyond this anastomosis to reperfuse the reimplanted trunk. After isolating the second MCA branch between aneurysm clips and trimming the bypass graft to size, it is implanted on the second MCA trunk with an end-to-side anastomosis. The second MCA branch is occluded with a permanent aneurysm clip at its origin from the aneurysm, and the blood flow is initiated in the bypass graft to reperfuse this trunk. **(d)** This reconstruction requires three anastomoses and allows the aneurysm to be excluded from the circulation. (Reproduced with permission from Lawton MT, Quinones-Hinojosa A. Double reimplantation technique to reconstruct arterial bifurcations with giant aneurysms. Neurosurgery 2006;58(4, Suppl 2):ONS-347–353.)

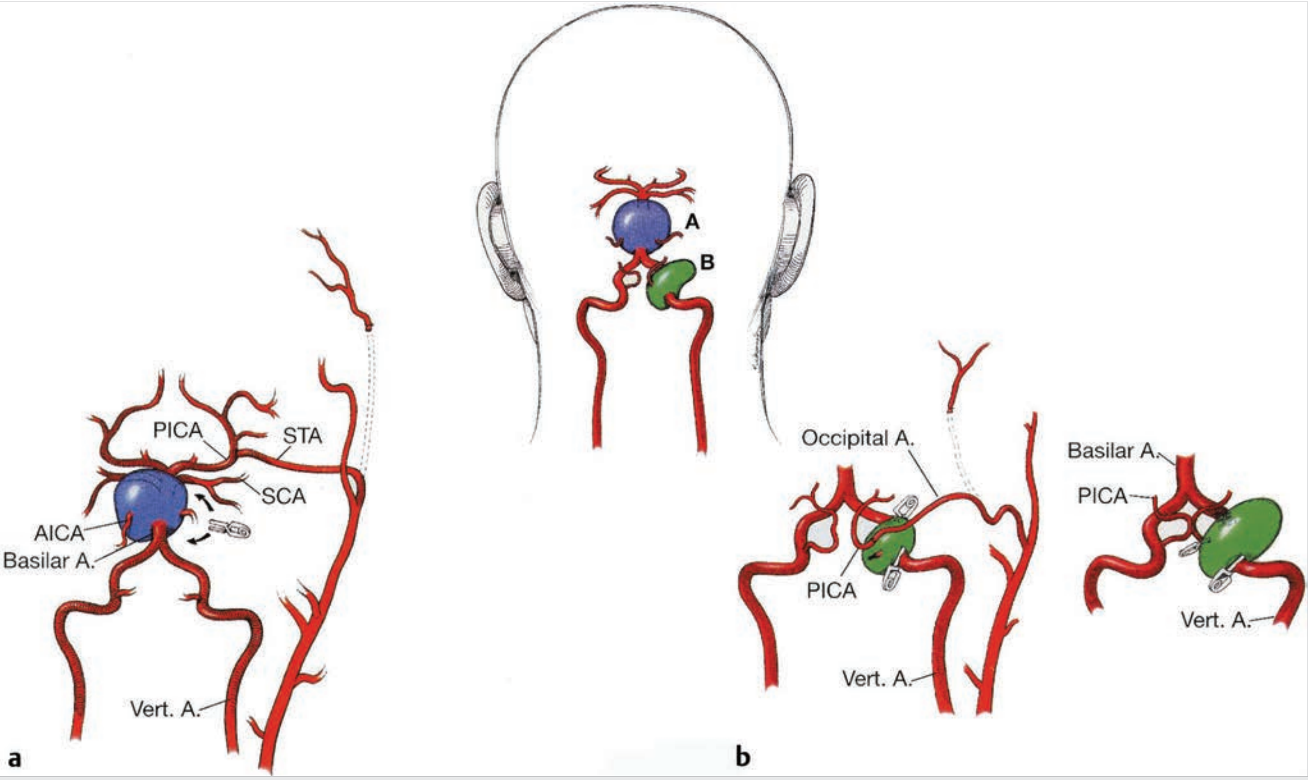

Fig. 18.7 Surgical approaches to revascularization of the posterior circulation. **(a)** The midbasilar artery is occluded proximally or distally to the aneurysm and revascularized with a superficial temporal artery (STA)-to-posterior cerebral artery bypass or an STA-to-superior cerebellar artery bypass. **(b)** The vertebral artery aneurysm is trapped (clips are placed on the proximal vertebral artery and at the origin of the posterior inferior cerebellar artery [PICA], and endovascular coils are placed distally in the vertebral artery) and revascularized with a PICA–PICA in situ bypass. Alternatively, an occipital artery-to-PICA bypass is shown. SCA, superior cerebellar artery; AICA, anterior inferior cerebellar artery; Vert. A., vertebral artery. (Reproduced with permission from the Barrow Neurological Institute, Phoenix, AZ.)

is wrapped circumferentially around the artery, and secured with an angled clip to constrict the sling against the vessel wall.

18.4.8 Endovascular Treatment

In cases of dissection requiring simple proximal occlusion, or trapping of a short arterial segment without perforating or branch arteries, endovascular embolization of the parent artery can be considered as an alternative to open surgery. Flow-diverting stents may offer an alternative to microsurgical revascularization for fusiform aneurysms, potentially re-establishing a normal arterial lumen while preserving perforating branches.[6] Data on safety and efficacy of these devices for fusiform aneurysms of the anterior circulation remains mixed and limited to small case series. Small case series for basilar artery fusiform aneurysms suggest that thrombotic and hemorrhagic complications with flow-diverting stents may exceed the natural history. In the setting of SAH, the aggressive antiplatelet therapy necessitated by flow diversion devices is associated with significant hemorrhagic complications, rendering stenting an option of last resort with ruptured aneurysms.

18.5 Postoperative Management Including Possible Complications

All patients are observed in a neurosurgical intensive care unit overnight. Aside from the standard postoperative measures necessary following a craniotomy, all bypass patients begin taking aspirin on postoperative day 1. All patients undergo an angiogram on postoperative day 1 to assess graft patency and aneurysm obliteration. In the case of dissecting aneurysms, any suggestion of postoperative residual should be followed closely with short interval angiography. Daily Doppler ultrasound studies can be useful for assessing bypass patency. If there are complications and postoperative neurological deficits, they typically become apparent immediately upon the patient's awakening. Most graft occlusions occur within the first 24 hours after surgery, and bypasses that are patent at that time have remained patent. Patients who develop graft occlusion and stroke should be considered for early decompressive hemicraniectomy, particularly in the setting of SAH or large residual aneurysm thrombus. Empirical antiepileptic medication is not used postoperatively, unless the patient had documented seizures preoperatively.

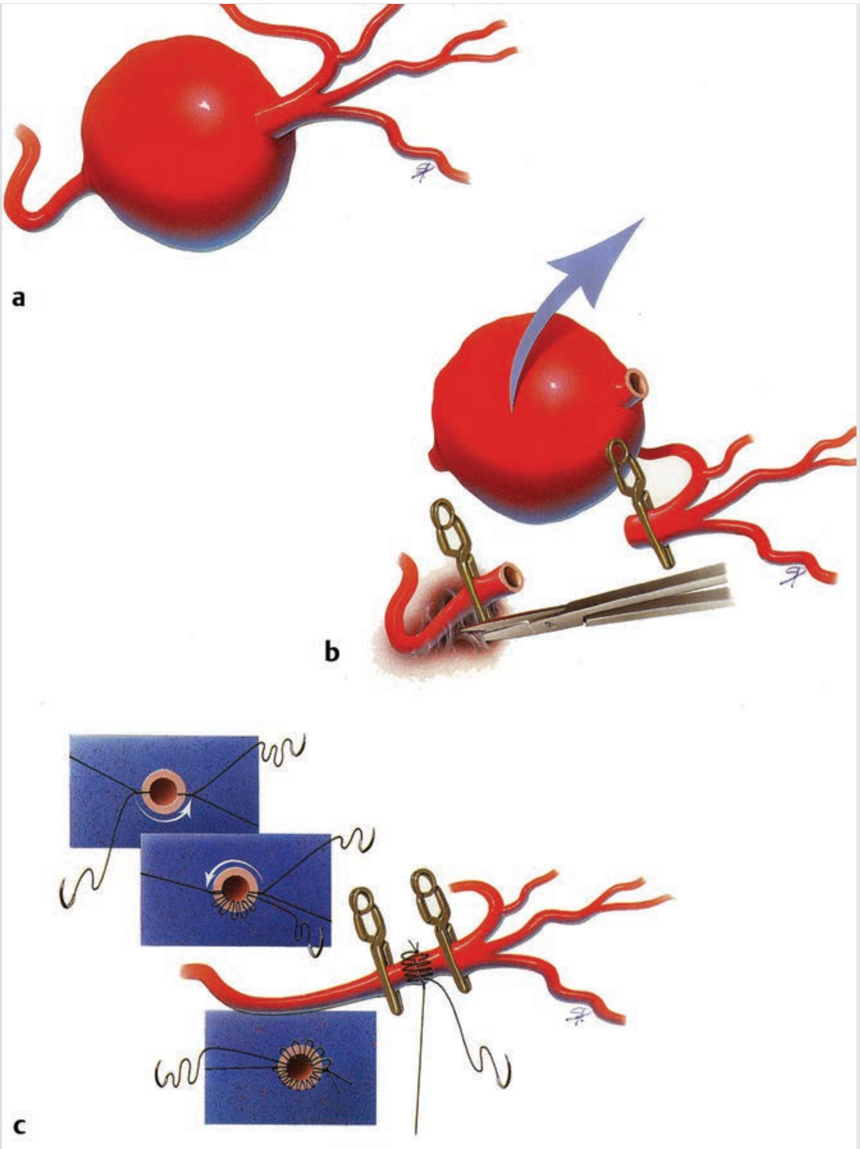

Fig. 18.8 **(a)** This proximal middle cerebral artery (MCA) dolichoectatic aneurysm is amenable to resection with primary reanastomosis. **(b)** Proximal and distal control of the aneurysm is obtained with temporary clips, and the aneurysm is resected. The ends of the artery are mobilized by cutting their arachnoid adhesions. **(c)** The proximal and distal ends are reapproximated with sutures placed opposite to each other. A continuous suture is run 180 degrees to the opposite suture; the stitches are not tightened until all are in place. The suture is then tightened and tied. In a similar fashion, the other half of the anastomosis is completed and the temporary clips are removed. (Reproduced with permission from the Barrow Neurological Institute, Phoenix, AZ.)

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19 Endoscopic Approaches to Intracranial Aneurysms

Alexander M. Tucker, Sergei Terterov, and John G. Frazee

Abstract

The use of endoscopy is increasing in neurosurgery. This exemplifies the trend toward minimally invasive approaches, and subsequently endoscopes have become widely accessible. With proper training, future vascular neurosurgeons will be required to perform safe and effective aneurysm surgery using the endoscope through smaller craniotomies with improved visualization.

Keywords: aneurysm, endoscopic surgery

19.1 Introduction

The use of the neuroendoscope has increased in recent decades in the treatment of many cranial pathologies. In particular, endoscopic procedures are now preferred by many surgeons when treating lesions of the sella turcica and anterior skull base. While many aneurysms today are amenable to endovascular treatment, some are still only treatable with surgical clipping. In the past, the adoption of the endoscope to treat intracranial aneurysms has been slow due to a steep learning curve, lack of instrument availability, and the large size and length of most endoscopes. Many of the traditional neuroendoscopes are 14 cm in length and 4 mm in diameter, making them unwieldy. In more recent years, there has been a trend toward minimally invasive approaches, and subsequently endoscopes have become widely accessible. With proper training, future vascular neurosurgeons will be required to perform safe and effective aneurysm surgery using the endoscope through smaller craniotomies with improved visualization.

Critics of endoscope-assisted aneurysm clipping argue that the camera limits the operator to a monocular view and only permits one-handed surgery. Further, some believe that in cases of aneurysm rupture, endoscopy dangerously narrows the field of view and compromises patient safety. A similar argument was made five decades ago against the use of the intraoperative microscope. However, with increased use and increasing surgeon comfort, the intraoperative microscope is now considered essential in the open treatment of cerebral aneurysms.

The most important step in applying the endoscope to aneurysm surgery is the design of a small, short, lightweight scope and camera (▶ Fig. 19.1). The Storz Image 1 camera body has been combined with an 11-cm long rigid glass scope with a lens diameter of 4 mm. Permanently attached to the camera body is a lightweight cable, which is connected to the camera box. A removable light cable plugs into the camera body just below the electronics cable. It is easily removed for sterilization. The total weight the surgeon holds is just 219 g and includes the camera, cords, scope, and suction. The camera body has, at the end opposite the cables, a bayonet mount for attaching the endoscope. There is a focusing wheel on the body and three push buttons for normal camera functions such as white balance. By adding to this endoscope an adjustable length suction of various diameters, which slides over the endoscope shaft, the surgeon can perform the surgery with two hands very much as he normally would work with a suction in one hand and an instrument in the other. The value of a high-definition and stereoscopic picture is overridden by placing the endoscope close to the anatomy of interest. Because this endoscope system is so lightweight and the attached cables do not drag, there is no significant hand fatigue from holding the endoscope.

Given its minimally invasive nature, endoscopic aneurysm surgery combines the advantages of definitive open surgery with the limited morbidity of endovascular treatment. Traditional endoscopic surgery (usually performed in the ventricular

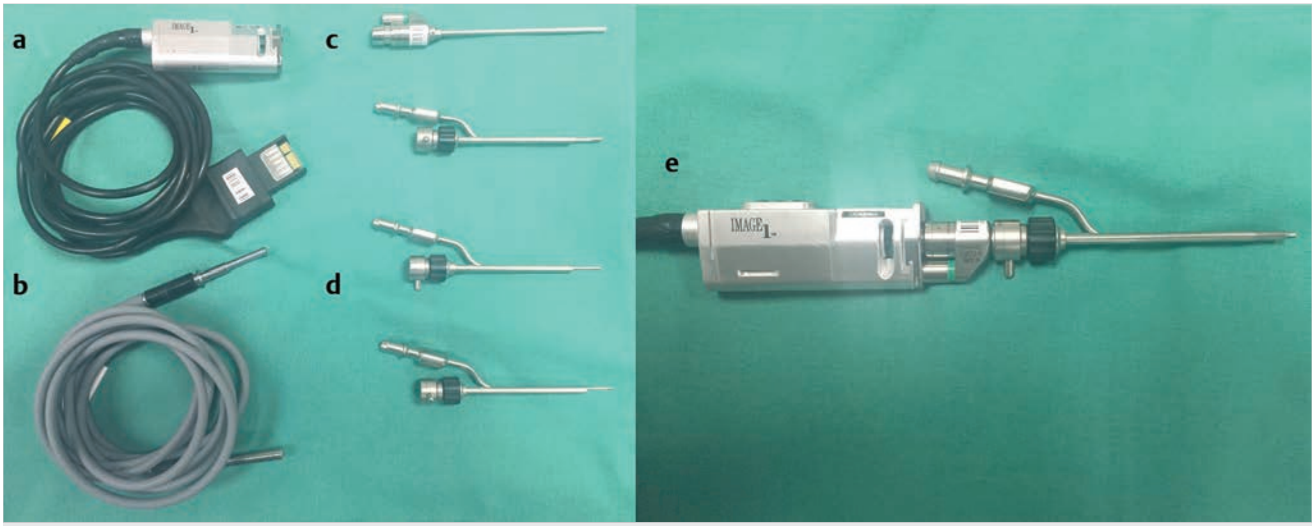

Fig. 19.1 Storz Frazee II Mini-endoscope. **(a)** Image 1 camera (8.4-cm long, 1.9-cm wide, and 3.3-cm tall, 98 g). **(b)** Cable from light source to camera (16 g). **(c)** 0-degree endoscope (4-mm diameter, 10.6-cm long) **(d)** Suction tips in three sizes (1.9-mm, 2.4-mm, and 3.3-mm in diameter). The length of each suction tip is adjustable (from 1.9 to 10 mm beyond the tip of the scope). **(e)** Fully assembled Frazee II Mini-endoscope (total weight 219 g).

system) uses an endoscope sheath through which irrigation and instruments are passed. However, in endoscope-directed surgery, the surgeon's primary operative instrument is passed alongside or parallel to the camera. In these cases, there is no role for the intraoperative microscope as visualization is as good or better with the hand-held endoscope. This technique is distinguished from endoscope-assisted surgery where the camera is merely used as an adjunctive tool to the microscope for close examination of the surgical bed or to look behind the aneurysm in areas obscured from the direct view of the operator.

There are many clear advantages to endoscopically directed surgery including smaller craniotomies (as small as 15 mm), minimal brain retraction, reduced manipulation of the aneurysm before clipping, better visualization of perforating arteries, and improved assessment of the surgical clip placement. Compared with the intraoperative microscope, the neuroendoscope offers superior brightness, increased depth of field, up to 25% greater magnification, a much closer view of the anatomy, and better maneuverability. These advantages highlight many of the shortcomings of the microscope including the need for generous surgical openings to permit direct lines of sight to all important anatomic structures relevant to the particular procedure.

19.2 Patient Selection

Endoscopic techniques can be used for both ruptured and unruptured aneurysms. In cases of acute rupture, most neurosurgeons advocate treatment within the first 24 hours to reduce the risk of rerupture. Given the increasing role of endovascular therapy, the treatment strategy for each aneurysm is formulated on a case-by-case basis after discussion with the interventional neuroradiology team.

19.3 Preoperative Preparation

After the decision is made to operate on a patient, consideration is then given to the potential role of the endoscope. Initially, the surgical team should evaluate if the aneurysm is approachable and treatable with the endoscope alone. If it is determined that the aneurysm cannot be wholly treated in an endoscopic fashion, then the endoscope may be used to assess the aneurysm anatomy and the regional vascular anatomy and to verify accurate clip placement (endoscopically assisted surgery). Many aneurysms, particularly posterior communicating, anterior communicating, middle cerebral bifurcation, and internal carotid artery bifurcation aneurysms, can be treated using the endoscope as the only means of viewing and completing the surgery (endoscopically directed surgery). Even basilar tip and superior cerebellar artery aneurysms can be treated in this manner. Distal aneurysms of the middle cerebral artery are generally not candidates for endoscopically directed surgery at present. However, pericallosal aneurysms can be approached with the endoscope with or without the use of image guidance.

In most cases, computed tomography angiography alone is the only preoperative vascular imaging necessary. It provides three-dimensional imaging and a fly-through tour from outside the skull through the cranial opening to the aneurysm. In some cases, digital subtraction angiography is necessary to evaluate aneurysm morphology or critical vascular anatomy, when the computed tomography angiography is of poor quality or the aneurysm anatomy requires time-resolved imaging.

19.4 Operative Procedure

19.4.1 Preparation and Positioning

All patients undergoing surgical treatment of aneurysms should be placed under general anesthesia with endotracheal intubation. At the start of induction, mannitol (1 g/kg) can be administered intravenously to enhance brain relaxation, thereby reducing the need for brain retraction. The timing of mannitol administration in endoscopically directed surgery is important and this should be communicated to the anesthesia team. Because smaller craniotomies can be used, it often takes 15 minutes or less from skin incision to dural opening and mannitol often is given sooner than would be expected for traditional open procedures. The authors administer antiepileptic medication at the start of the operation. Placement of an external ventricular drain is performed before surgery in most cases of ruptured aneurysms, but is usually unnecessary when treating unruptured aneurysms. If an external ventricular drain is not placed, further brain relaxation can be achieved by opening the cerebrospinal fluid (CSF) cisterns as well as by fenestrating the lamina terminalis. This technique cannot be used prior to clipping in cases of anterior communicating artery aneurysms. In these cases, if the brain seems tight, an external ventricular drain can be placed before clipping.

Many aneurysms of the anterior circulation can be approached through a small eyebrow incision (▶ Fig. 19.2). The patient's head is placed in a neutral position facing the ceiling. Then, the head is slightly extended to allow the frontal lobes to fall away from the anterior fossa floor. The supraorbital notch should be palpated and the skin incision marked approximately 2.5 cm laterally to this point to avoid injuring the exiting supraorbital nerve. In order to improve cosmesis, the incision can be placed just under the lower margin of the eyebrow and the hair left unclipped.

For aneurysms of the posterior circulation or when an eyebrow approach is not indicated, the patient should be positioned in a similar manner to more traditional open aneurysm clipping cases. For basilar tip and superior cerebellar aneurysms, a subtemporal approach is used with a similar-sized incision and bony opening. In endoscopic-directed surgery, the skin incision is typically so small that only a small patch of hair, about 1-cm wide, is clipped for the distance of the skin incision, what we have come to call the "Hollywood shave." After surgery, the remaining hair can be combed over the path to hide the incision.

Many techniques have been used to preserve the patency of adjacent perforating arteries and the parent vessel, including monitoring evoked potentials and electroencephalography, intraoperative angiography, fluorescent microscopy, Doppler ultrasonography, and endoscopy. For most cases of endoscopic aneurysm clipping, we use somatosensory evoked potentials and electroencephalography as well as intraoperative Doppler

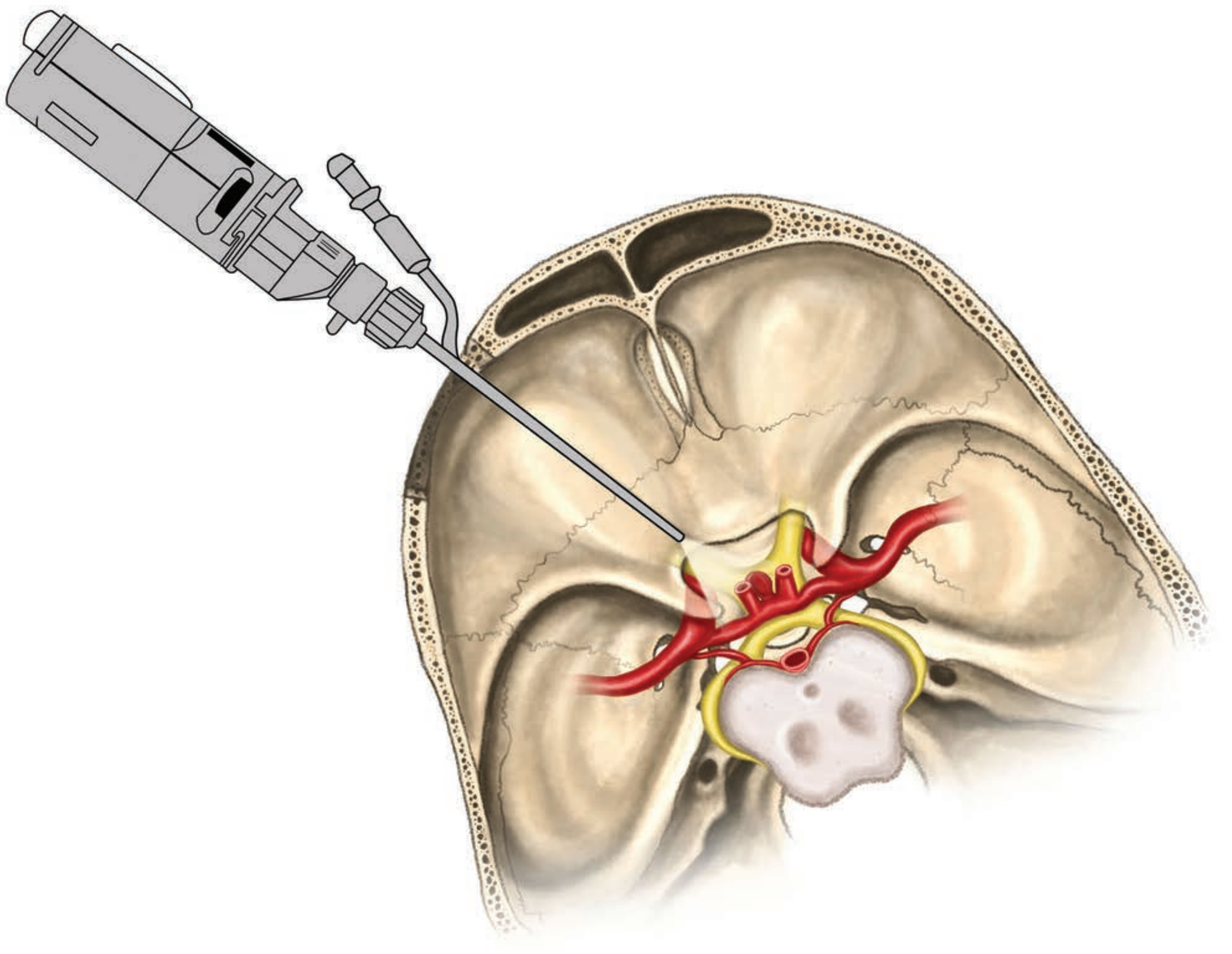

Fig. 19.2 Approach to anterior communicating artery aneurysm. An eyebrow incision and a subfrontal approach along the floor of the anterior fossa can be used to surgically access most anterior circulation aneurysms.

ultrasound. We believe the high-resolution view of the clip construction obviates the need for intraoperative angiography.

19.4.2 Surgical Opening

After prepping and draping in the standard fashion, the skin is incised to the level of the skull. The scalp is undermined in all directions. Superficial soft tissue is retracted with fish hooks or a small self-retaining retractor. For the eyebrow approach, a single burr hole is made in the most lateral aspect of the exposure. A small craniotomy is created, approximately 2.5-cm wide and 1.5-cm high. With experience, for select cases, it may be possible to use an even smaller bony exposure. The dura is opened in a **U**-shape with its pedicle located inferiorly. This should be carefully tacked back and out of the operator's field of view (▶ Fig. 19.3). With proper positioning, the frontal lobes should relax away from the anterior fossa floor. This relaxation is aided by CSF egress after opening the arachnoid. In some cases when the effect of gravity on the brain is insufficient, a self-retaining malleable brain retractor is used to elevate the frontal lobes. Otherwise, the 4-mm 0-degree rigid glass Frazee II mini-endoscope is placed in the subdural space and further CSF is removed with suction.

19.4.3 Operation of the Neuroendoscope

For right-handed neurosurgeons, the endoscope is best held in the left hand as one would hold a suction cannula. This generally allows the operating surgeon to use instruments in both hands, the endoscope and suction in the left and the dissecting, coagulating, or clipping instruments in the right. Because the endoscope is so lightweight, no scope holder is necessary. The surgeon has the freedom to move the endoscope in all directions in order to visualize the important anatomy. By positioning the endoscope close to the area of interest and by frequently changing the viewing angle, the operator can achieve a detailed, three-dimensional appreciation of critical brain structures. Frequent repositioning of the mini-endoscope can mitigate concerns about the lack of depth perception when conducting surgery through the endoscope. Movement of the mini-endoscope must be done

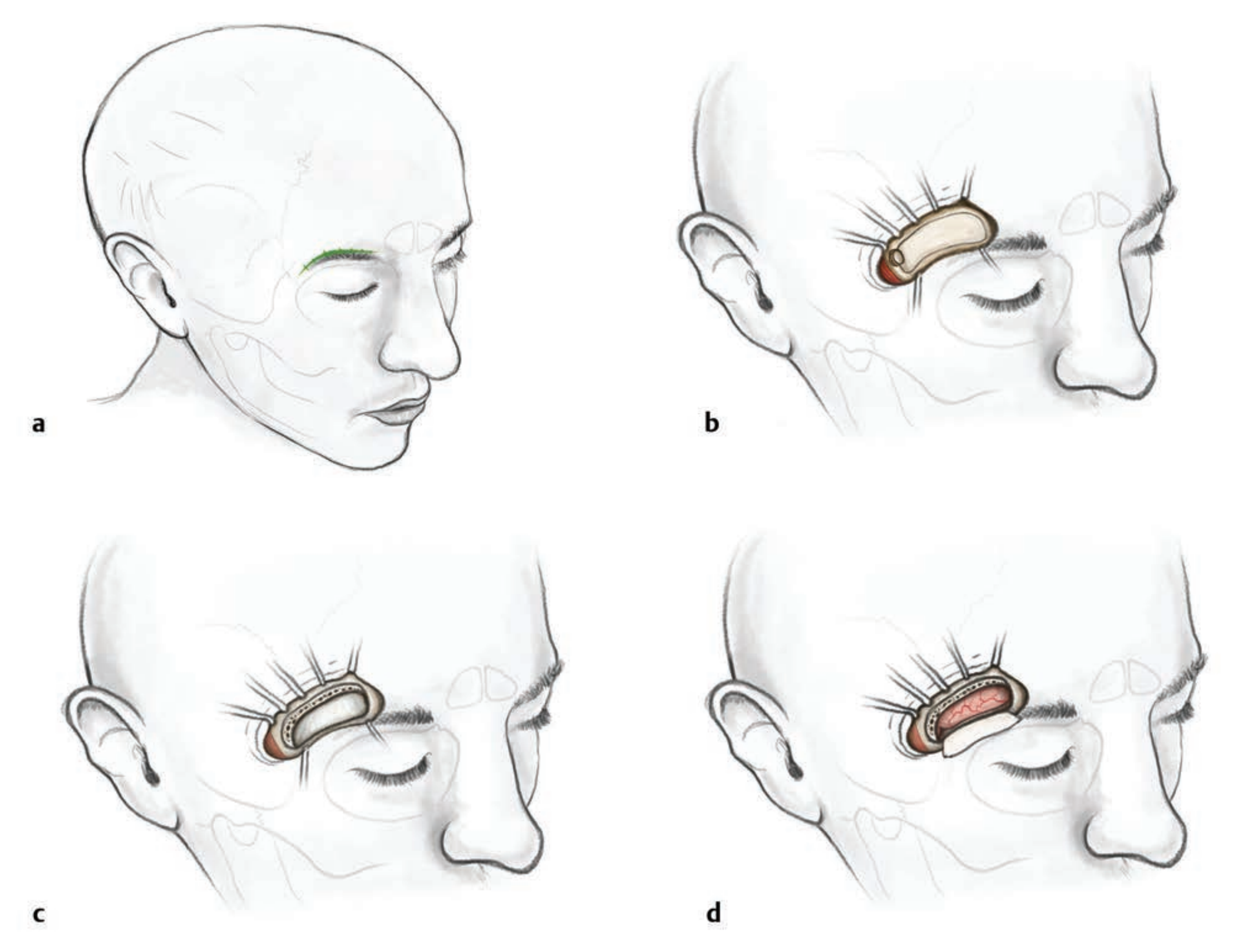

Fig. 19.3 Eyebrow approach. **(a)** Skin incision is made just below the eyebrow, lateral to the supraorbital notch. **(b)** Skin is reflected with fish hooks, temporalis dissected away from the skull, and keyhole identified. **(c)** A single burr hole is placed at the keyhole and a 2.5 cm × 1.5 cm craniotomy is elevated. **(d)** Dura is opened in a **U** shape with the pedicle oriented inferiorly.

slowly and carefully to prevent damage to neural and vascular structures. Furthermore, it is critical for the surgeon "driving" the endoscope to recognize that delicate structures often lie along the shaft of the scope and are vulnerable to injury with movement, but are outside the field of view.

Some neurosurgeons advocate the use of angled endoscopes. Although these endoscopes can provide better visualization of some hard-to-see areas, caution should be used before choosing a camera with a high-angled lens. Lenses of 30, 70, or even 120 degrees are available and useful but can provide a confusing picture of the operative site to neurosurgeons unfamiliar with endoscopy. For most aneurysms, careful preoperative planning allows the surgery to be conducted via a direct path to area of pathology. For these cases, a rigid, 0-degree endoscope, such as the Frazee II mini-endoscope, is almost always preferred and sufficient.

19.4.4 Surgical Technique for Anterior Circulation Aneurysm Clipping

When performing endoscope-directed surgery, the operator must adhere to the principles of aneurysm surgery. These include exposure of the proximal feeding artery for potential temporary clipping, beginning dissection at the neck of the aneurysm, visualizing all perforating arteries and branches prior to clip placement, and careful inspection of the clip construct after the aneurysm is treated. Of particular note, when treating posterior communicating artery aneurysms closely adherent to the oculomotor nerve, the surgical clip should be placed without retracting or pulling on the dome of the aneurysm to avoid postoperative cranial nerve palsy.

Using the eyebrow incision, the olfactory tract is easily identified and then followed posteriorly to the lateral aspect of the optic nerve. The arachnoid over the optic nerve is then opened sharply and CSF is suctioned to allow for further brain relaxation. By further opening the arachnoid lateral to the optic nerve, the carotid artery is identified. At this stage, the carotid artery can be dissected circumferentially to allow for proximal control. The carotid artery is then followed distally until the posterior communicating artery is identified. If this is the location of the aneurysm being treated, the aneurysm neck is dissected. If the aneurysm is more distally located, dissection along the carotid artery is continued to the level of the carotid bifurcation. Anterior communicating artery aneurysms are approached by following the first segment of the anterior cerebral artery medially from the carotid bifurcation. If needed, the gyrus rectus can be subpially resected to better expose the aneurysm and other critical vascular structures. At this step, the recurrent artery of Huebner must be identified and preserved. Middle cerebral aneurysms can be identified by following the proximal middle cerebral artery distally, opening the arachnoid as needed.

Once the aneurysm has been identified, the neck is carefully isolated. A small dissector is placed between each of the branching arteries and the aneurysm neck after which an aneurysm clip is applied. In cases where complete circumferential visualization of the neck is not possible, a clip is applied across the visible aneurysm neck and the rest of the aneurysm is rapidly dissected. The dome of the aneurysm is often decompressed to facilitate better visualization. If an arterial branch is included in the clip blades, that artery is quickly dissected. A second clip, which excludes the branch, is applied to the aneurysm neck and the first clip removed or repositioned. In some cases, the bulk of the aneurysm obscures adequate visualization of relevant structures, particularly branching arteries. Bipolar cautery can be used on very low power (usually 20 on the Spetzler-Malis bipolar setting), to shrink the aneurysm dome and improve the surgeon's view. Care should be used to avoid pushing the sharp bipolar tips into the fragile aneurysm; instead, the bipolar tips should be held parallel to the dome of the aneurysm and gently touched to its surface. As this maneuver involves manipulation of the delicate dome, the aneurysm neck should be completely dissected and a surgical clip loaded in the applier in case of premature rupture. Keeping the aneurysm moist prevents the bipolar from sticking to the aneurysm.

19.4.5 Bleeding

In cases of unexpected aneurysm rupture, the technique is similar to cases of open surgery using the operative microscope. The endoscope should be pulled back and rinsed clean by the assistant. A large bore suction is placed near the aneurysm to help control the bleeding using the right hand, until control is reestablished with the suction attached to the mini-endoscope. If visualization is still challenging, then the suction may be moved immediately over the rupture site. Occasionally, a small cotton pledget must be used to tamponade the bleeding. By gently compressing the rupture site for approximately 1 minute, bleeding usually can be controlled and dissection may continue. Alternately, in some cases dissection can continue concomitantly while pressure is being held over area of bleeding. Occasionally, a small brain retractor on a flexible arm has been used to hold a pledget on the aneurysm bleeding site, tamponading the bleeding and freeing the surgeon's hands. If bleeding is not controlled with these techniques, a temporary clip may be placed on the aneurysm or proximal artery until the dissection is completed and a permanent clip can be optimally placed. At the conclusion of the surgery, the lamina terminalis is opened if there has been a subarachnoid hemorrhage.

19.4.6 Closure

After definitive clipping of the aneurysm is completed, the surgical field should be copiously irrigated until clear. Arteries are bathed in papaverine solution and the endoscope is gently withdrawn. Retractors, if used, are removed and underlying brain carefully examined for injury. The dura is closed with 4–0 suture and the original bone flap secured into place with low-profile titanium plates and screws. The temporalis, if dissected, and the galea are reapproximated with 3–0 absorbable suture. Finally, the skin is closed with 4–0 absorbable suture if in a visible location or with surgical staples if behind the hairline. A sterile wrap is then tightly placed around the head for 24 to 48 hours to reduce any scalp bleeding and periorbital swelling.

19.5 Postoperative Management Including Possible Complications

Patients with aneurysms treated via an endoscopically directed approach should be managed in a similar manner to those treated in the traditional fashion. Prophylactic antibiotics are continued for one postoperative dose and steroids, if given at the start of surgery, are rapidly tapered off over 1 week. In cases of preoperative or intraoperative aneurysm rupture, antiepileptic medications are continued for a minimum of 2 weeks, although this course may be extended if the patient suffers a seizure. Postoperative pain is typically minimal and is treated with intravenous narcotics, usually morphine. When the patient is able to take enteral medication, primary pain control is achieved with narcotics, usually oxycodone and acetaminophen. Patients with subarachnoid hemorrhage are kept in the intensive care unit for 2 to 3 weeks for frequent neurologic examination and blood pressure management with a central venous catheter in place. Daily transcranial Doppler ultrasound is used to monitor intracranial arterial velocities. If elevated velocities are detected that suggest angiographic vasospasm, then hypertensive therapy and volume loading are used. Systolic blood pressures of 180 to 200 mm Hg are commonly reached using intravenous vasopressors to treat or prevent symptomatic vasospasm. Of note, treating vasospasm like this is only appropriate in cases when the aneurysm has been completely secured. For severe vasospasm or when the aneurysm was only partially secured during surgery, catheter angiography with verapamil injection and rarely angioplasty is required.

External ventricular drainage is used is many patients, particularly those with evidence of acute hydrocephalus on presentation, in patients with intraventricular hemorrhage, or in those with suspected increased intracranial pressure. Some patients will develop chronic hydrocephalus, although the likelihood of this developing is greatly decreased when intraoperative third ventriculostomy through the lamina terminalis is performed. If the external ventricular drain cannot be weaned postoperatively, then these patients are candidates for a CSF diversion procedure. As all cases of subarachnoid hemorrhage have had the lamina terminalis opened, the procedure of choice for those patients developing hydrocephalus is placing a ventriculoperitoneal shunt with an antibiotic-impregnated catheter and a programmable valve. Ventriculoperitoneal shunts are best for patients with significant intraventricular hemorrhage. Similarly, shunting is preferred and endoscopic ventriculostomy should be avoided when there is a history of CSF infection as this causes high fenestration failure rates.

20 Microsurgical Treatment of Previously Endovascularly Treated Aneurysms

Badih Junior Daou, Nohra Chalouhi, Stavropoula Tjoumakaris, Pascal Jabbour, and Robert H. Rosenwasser

Abstract
Cerebral aneurysms are being managed more commonly using endovascular techniques. The positive short-term outcomes of endovascular therapy have been well established. However, there exists an increasing population of patients treated with endovascular management, especially coiling, and longer duration of follow-up. The long-term drawbacks, such as recurrent subarachnoid hemorrhage and persistent residual and regrowth of aneurysms, are increasingly evident despite initial endovascular treatment. Management of these rehemorrhaged/residual/recurrent aneurysms requires a challenging multitude of surgical and endovascular techniques.

Keywords: cerebral aneurysm, clipping, endovascular coiling, aneurysm recurrence

20.1 Patient Selection

Endovascular treatment of cerebral aneurysms is increasing in part because of the better outcomes for aneurysms that are being managed more commonly using endovascular techniques. The positive short-term outcomes of endovascular therapy have been well established. However, with the increasing population of patients being treated with endovascular management, especially coiling, and longer duration of follow-up, the long-term drawbacks have evolved.[1,2,3] These include an important number of recurrences and residuals, coil compaction and extrusion, and occurrence of subarachnoid hemorrhage (SAH) despite initial endovascular treatment. With the growing number of recurrent aneurysms, the need for retreatment is expanding. Treatment of these recurrences and remnants can be challenging, but a multitude of surgical and endovascular techniques are available. The main surgical treatment options include direct microsurgical clipping, aneurysm wrapping, or extracranial–intracranial bypass with parent vessel occlusion (▶ Fig. 20.1, ▶ Fig. 20.2, ▶ Fig. 20.3, ▶ Fig. 20.4, ▶ Fig. 20.5). Endovascular treatment methods include recoiling, stenting, stent-assisted coiling, or the use of flow diversion devices such as the pipeline embolization device. Although treatment with recoiling is usually attempted, up to 50% of cases may still require further treatment.[4] Furthermore, some recurrences and remnants cannot be managed with a second endovascular intervention because of complex anatomy or difficult endovascular access. Surgical clipping of intracranial aneurysms results in higher rates of complete obliteration as compared to endovascular coiling. However, clipping of previously endovascularly treated aneurysms is more technically challenging than surgical clipping of naïve aneurysms and has its own difficulties attributed to the previously placed coils or stents. However, with appropriate patient selection, the efficacy of the procedure is high and procedural risks are low. The decision to treat recurrent, previously endovascularly treated cerebral aneurysms with microsurgical clipping depends on patient and aneurysm characteristics. Important factors to weigh in during the patient selection process include the following: age less than 70 years; no significant comorbidities or well-controlled comorbidities; patient unreliable to return for follow-up imaging when a recurrence is diagnosed; no prior and current history of smoking; presentation with SAH from the recurrence or residual; patient preference for surgical intervention; wide aneurysm neck; a dome-to-neck ratio of less than 2:1; aneurysm location, with recurrences arising from the anterior communicating artery, the posterior communicating artery, and the middle cerebral artery being the best surgical candidates; aneurysm neck/base incorporating adjacent branching arteries; aneurysms that are less densely packed with coils leaving enough space for clip placement at the neck; and presence of a significant recurrence/residual following the endovascular intervention (> 40%) or progression during follow-up occlusion (▶ Fig. 20.1, ▶ Fig. 20.2, ▶ Fig. 20.3, ▶ Fig. 20.4, ▶ Fig. 20.5). Microsurgical clipping can be performed after one failed endovascular intervention or multiple interventions. Older patients with several comorbidities, aneurysms in the posterior circulation, unfavorable dome-to-neck ratio, and coils densely packed in the aneurysm neck are less likely to be candidates for surgical clipping.

20.2 Preoperative Preparation

All patients should be assessed for comorbidities that can influence the operative and postoperative course, including a thorough assessment of cardiac and pulmonary status. A comprehensive neurologic assessment should be performed as well. Despite recent advances in computed tomography (CT) angiography and magnetic resonance angiography, digital subtraction angiography (DSA) remains the standard technique to evaluate the cerebral vasculature. Three-dimensional reconstruction with rotational DSA can further provide a detailed anatomic and topographic evaluation of the cerebral vascular tree. This can help in assessing the shape of the aneurysm, its relation to the parent artery, its spatial relationship to surrounding vessels, dimensions of the aneurysm neck, the neck-to-dome ratio, distribution and packing density of the previously placed coils within the aneurysm and the space at the aneurysm neck that is free of coils for clip placement, any deformation of the parent arteries secondary to the aneurysm or coil mass, direction of the aneurysm projection, patency of communicating segment arteries, and accessibility of the proximal parent arteries for proximal vascular control during clipping.

20.3 Operative Procedure

The procedure is performed under general endotracheal anesthesia with the administration of pentobarbital, mannitol, and furosemide to aid in achieving cerebral relaxation, which can improve the visualization and access to the aneurysm for microsurgical clipping and decrease the force required for cerebral retraction. Furthermore, cerebrospinal fluid volume management with a

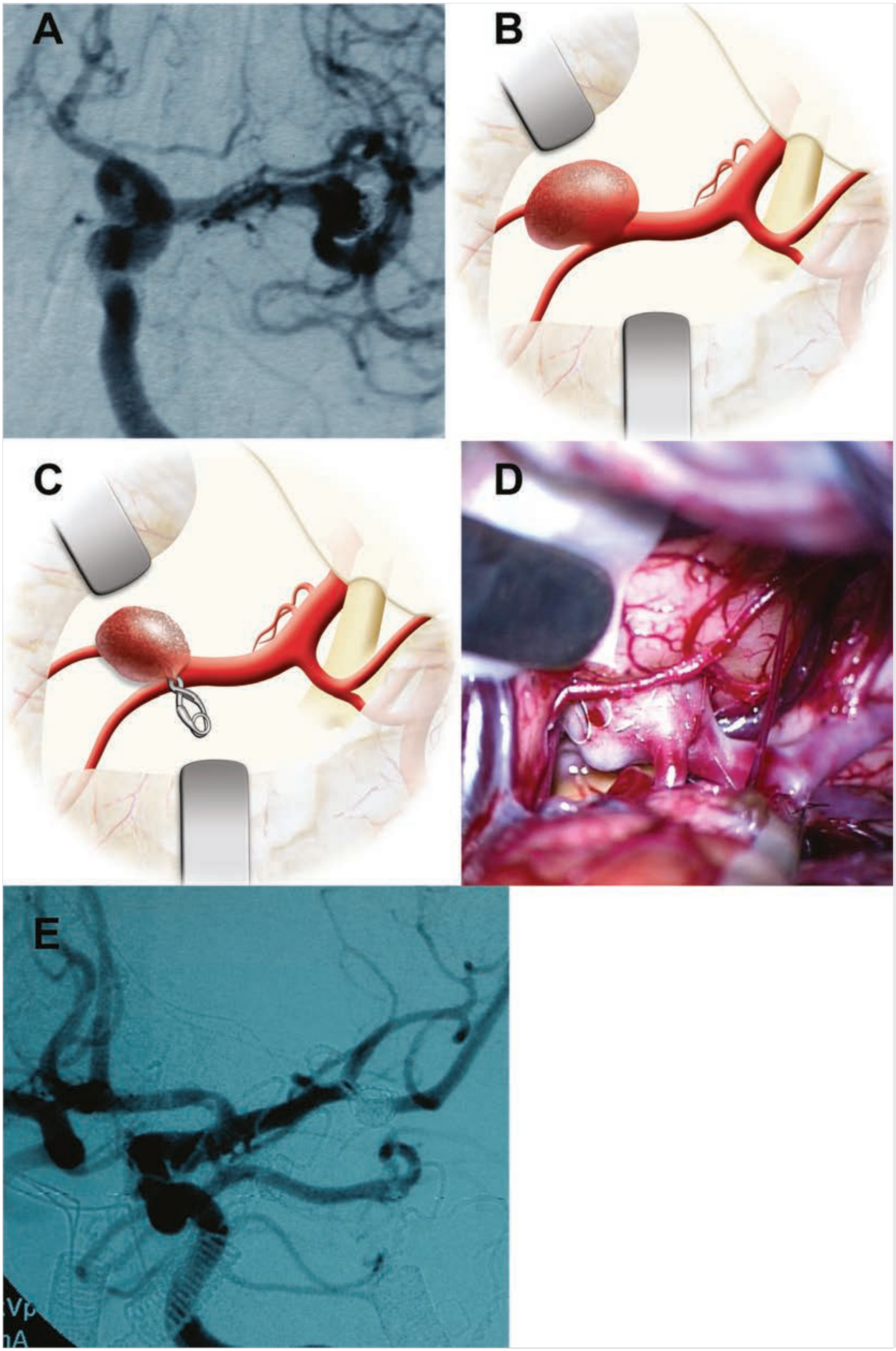

Fig. 20.1 **(a)** Anteroposterior left carotid angiogram showing a recurrent middle cerebral artery aneurysm after endovascular treatment with coils. **(b)** Drawing of the aneurysm exposed through a left pterional craniotomy with wide splitting of the sylvian fissure. **(c)** Intraoperative photograph of the aneurysm exposed through a left pterional craniotomy. The sylvian fissure has been opened and a retractor can be seen on the temporal lobe. **(d)** Drawing of the aneurysm after clip application. **(e)** Intraoperative carotid angiogram showing clipping of the aneurysm and patency of the middle cerebral artery and its branches.

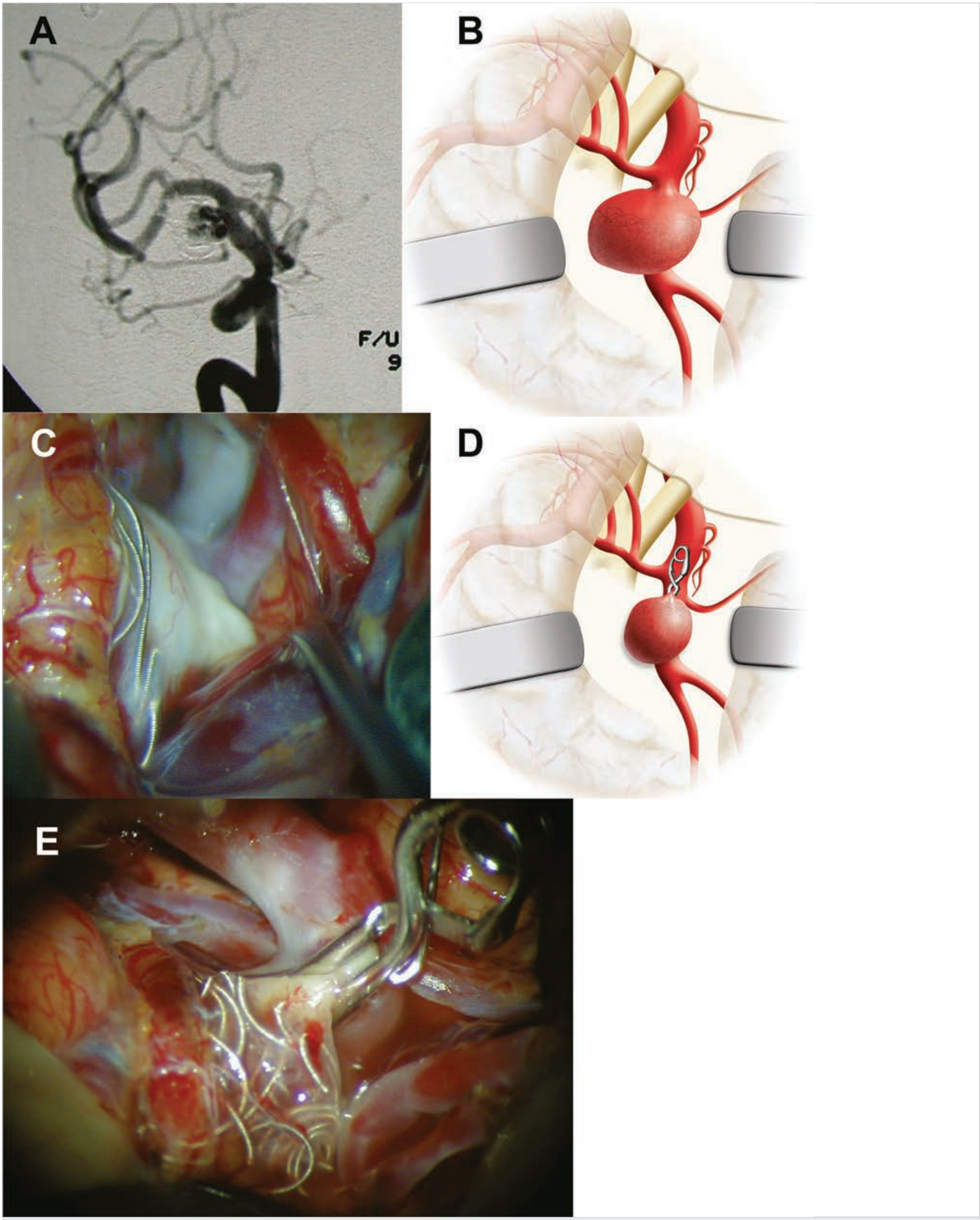

Fig. 20.2 (a) Anteroposterior right carotid angiogram showing a recurrent internal carotid artery aneurysm after endovascular treatment with coils. **(b)** Drawing of the aneurysm exposed through a right pterional craniotomy with wide splitting of the sylvian fissure. **(c)** Intraoperative photograph of the aneurysm exposed through a left pterional craniotomy. The sylvian fissure has been opened and a dissector can be seen retracting on the temporal lobe. **(d)** Drawing of the aneurysm after clip application. **(e)** Intraoperative photograph after clip application to the aneurysm neck.

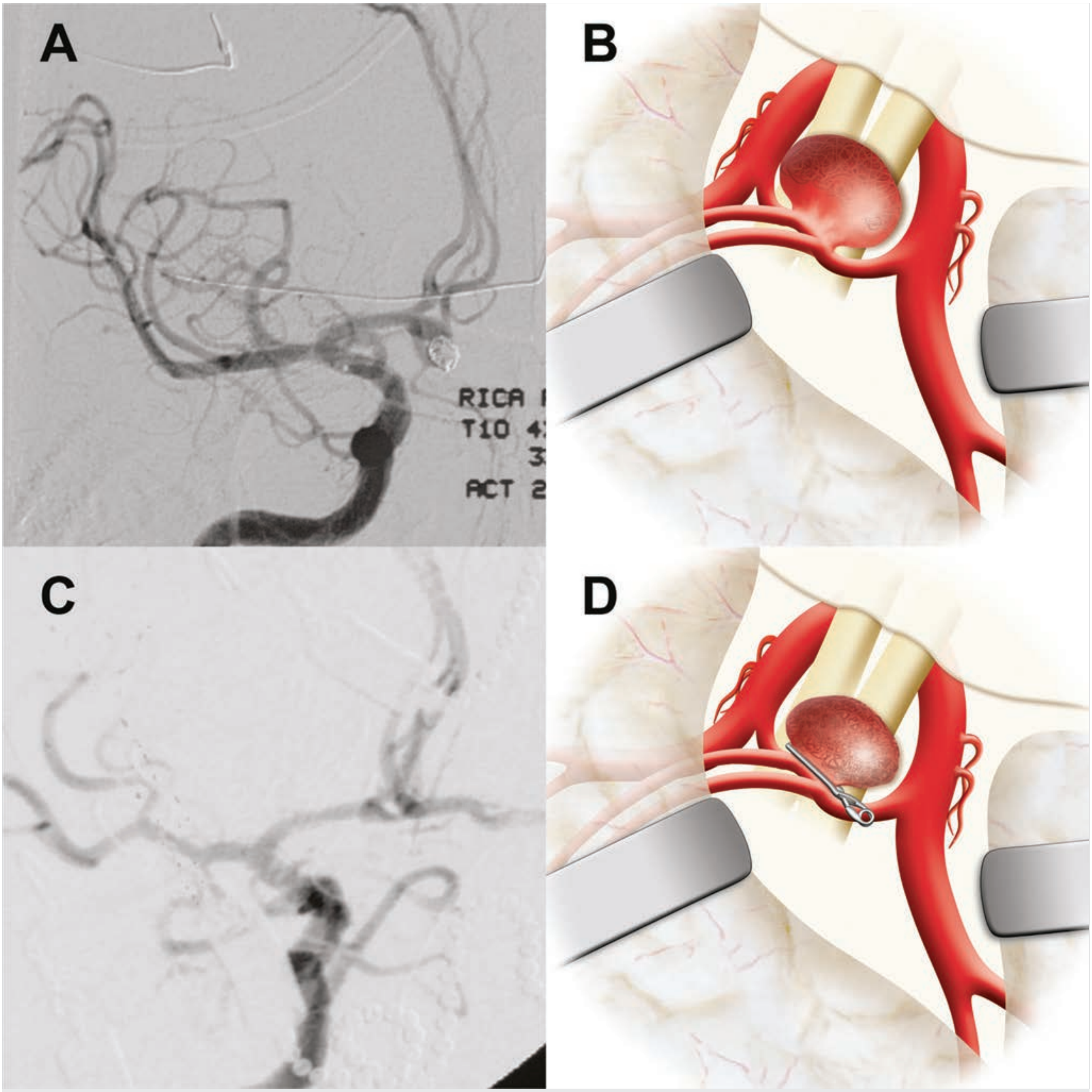

Fig. 20.3 **(a)** Oblique right internal carotid artery angiogram showing recurrent anterior communicating artery aneurysm after coiling. **(b)** Drawing of the previously coiled aneurysm exposed through a right pterional craniotomy. **(c)** Postoperative right internal carotid angiogram shows clipping of the aneurysm and patency of anterior cerebral arteries. **(d)** Drawing of intraoperative exposure of the aneurysm after clipping.

ventricular or lumbar drain can facilitate cerebral relaxation and reduce retraction on the cerebrum during the initial opening of the fissures and arachnoid spaces as well as improve cisternal exposure during the final stages of aneurysm exposure.

Neurophysiologic monitoring is commonly used intraoperatively and can reflect the effects of anesthesia, surgical manipulation, and changes in cerebral blood flow, and represents a valuable tool in preventing complications by allowing adjustments of surgical and anesthetic techniques. Continuous electroencephalography indirectly reflects the metabolic activity of the brain and can detect cerebral hypoxia and ischemia. Somatosensory and motor evoked potentials are useful to watch for impaired neurological function, including vessel occlusion, compromise to small perforating vessels, disturbed local microcirculation, and changes in cerebral perfusion. Brainstem auditory evoked potentials can be used during clipping of posterior circulation aneurysms and are highly sensitive in detecting brainstem ischemia. When neurophysiologic monitoring is performed, the anesthesiology and neurophysiology teams should work together to establish appropriate and rapid intraoperative management.

Technical difficulties attributed to the coils present in the aneurysmal sac and neck are frequently seen, especially with

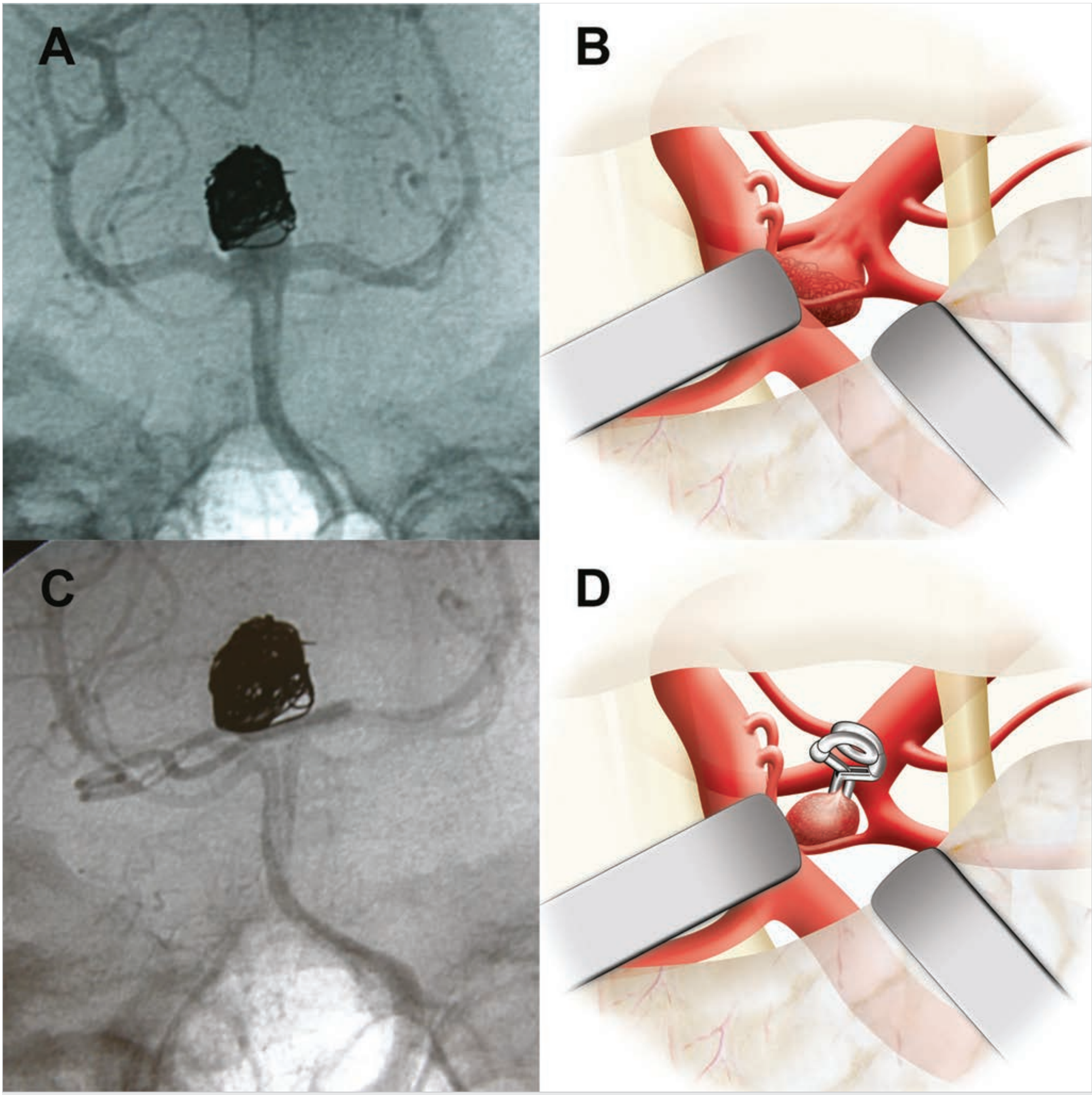

Fig. 20.4 **(a)** Anteroposterior vertebral artery angiogram showing recurrent basilar bifurcation aneurysm after coiling. **(b)** Drawing of the previously coiled aneurysm exposed through a right pterional craniotomy. **(c)** Postoperative anteroposterior vertebral artery angiogram shows clipping of the aneurysm and patency of posterior cerebral arteries. **(d)** Drawing of intraoperative exposure of the aneurysm after clipping.

the presence of a large coil mass that can make the process of clip placement around the aneurysm neck more cumbersome. For this reason, adequate exposure should be obtained and the craniotomy should be extended sufficiently with additional removal of the skull base to optimize the surgical space and allow adequate exposure to the aneurysm and its parent arteries, to further minimize cerebral retraction and allow the performance of more complex maneuvers that are often required with microsurgical management of previously endovascularly treated aneurysms.

20.3.1 Craniotomy and Skull Base Exposure

Several surgical approaches can be performed based on the location of the lesion. In general, a standard pterional craniotomy is adequate for managing aneurysms located in the anterior circulation with minimal brain retraction. Skull base techniques such as a modified orbitozygomatic craniotomy or a tailored orbitotomy can further provide a larger operative field and less brain retraction especially in the case of large, complex anterior

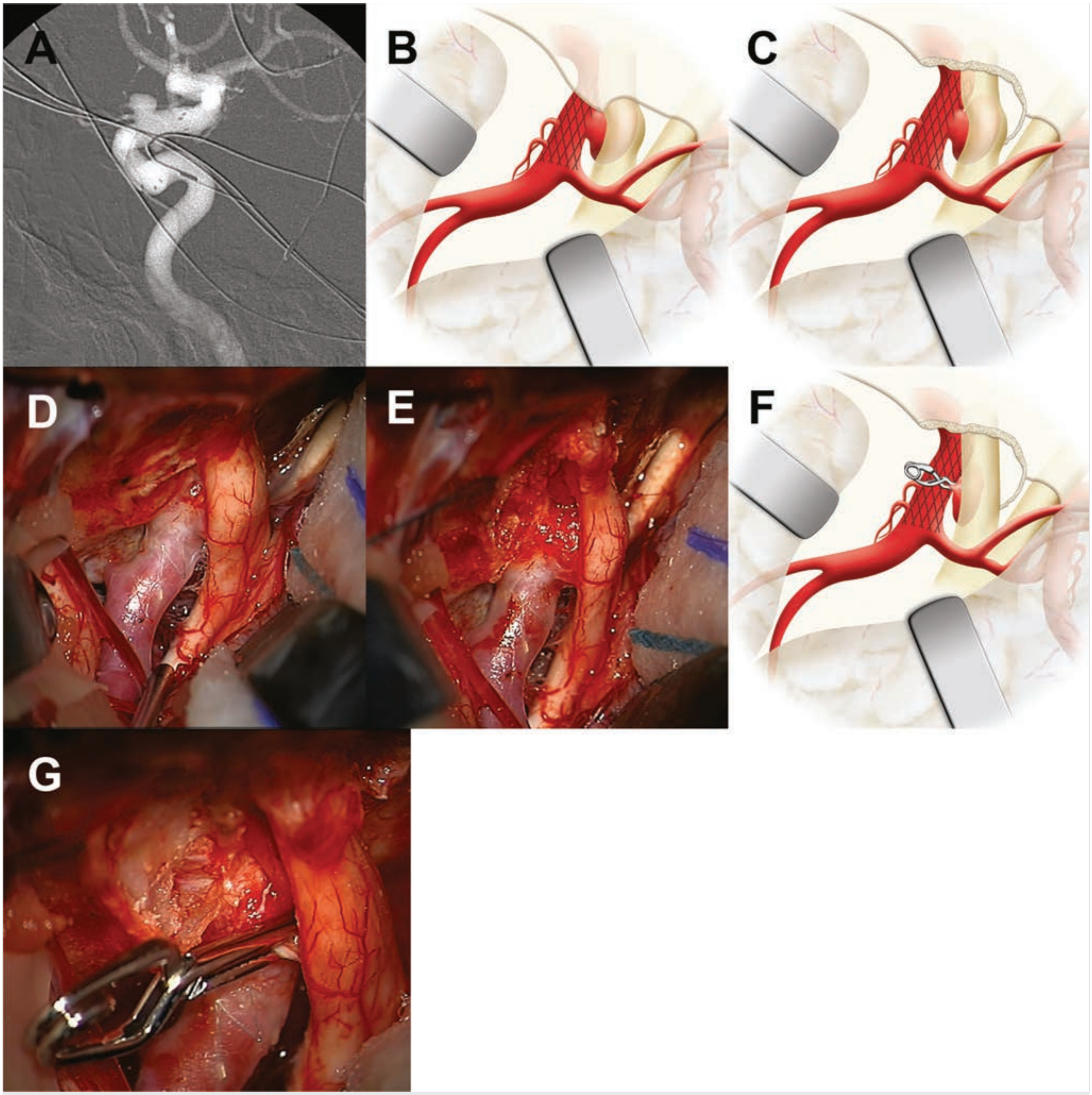

Fig. 20.5 **(a)** Lateral left internal carotid artery angiogram showing a stent in the internal carotid artery that spans across the neck of an ophthalmic artery aneurysm. **(b)** Drawing of the aneurysm exposed through a left pterional craniotomy before and **(c)** after the anterior clinoid process is drilled away. **(d)** Intraoperative photograph of the aneurysm exposed through a left pterional craniotomy before and **(e)** after drilling away the anterior clinoid process. **(f)** Drawing and **(g)** intraoperative photograph after clipping of the aneurysm.

communicating artery, posterior communicating artery, internal carotid artery cave, clinoidal, ophthalmic, superior hypophyseal, and internal carotid artery terminus aneurysms. Removal of the anterior clinoid process extra- or intradurally is necessary to facilitate exposure of clinoidal, ophthalmic, superior hypophyseal, and some posterior communicating aneurysms.

Various cranial base approaches can be performed in the management of posterior circulation aneurysms, including the far lateral suboccipital craniotomy for posterior inferior cerebellar and vertebral artery aneurysms, orbitozygomatic plus posterior clinoid resection or subtemporal route with anterior petrosectomy for basilar apex and superior cerebellar artery aneurysms, far lateral/retrosigmoid craniotomy for anterior inferior cerebellar artery and distal vertebral aneurysms, and petrosectomy for vertebrobasilar junction and basilar trunk aneurysms.

20.3.2 Microdissection

Splitting of the sylvian fissure can improve exposure to anterior circulation aneurysms, minimize cerebral retraction, and aid in brain relaxation especially in the presence of large and complex aneurysms. The venous anatomy on the surface of the sylvian fissure varies widely. Multiple large veins often follow the course of the sylvian fissure. Even when retractors are carefully placed, splitting the sylvian fissure and microsurgical dissection result in exposure of these venous structures. Division of these veins should be minimized since injury can lead to edema, hemorrhage, venous insufficiency, and, potentially, venous infarction. Opening the lamina terminalis can improve brain relaxation and visualization for clipping anterior communicating artery aneurysms.

Arachnoid dissection with opening of the basal cisterns also aids in minimizing brain retraction, improving brain relaxation, and improving exposure of the aneurysm. Opening the arachnoid at the cisterna magna with microscissors around cranial nerves VII/VIII, IX, X, XI, and XII with posterior lateral (retrosigmoid and far lateral) approaches and dividing the ipsilateral uppermost dentate ligament with the distal far lateral approach should be performed to avoid retraction on the brainstem and cranial nerves.

Previous endovascular management of the aneurysm may result in significant scarring, adhesions, and changes in the aneurysm morphology that may complicate the procedure. Sharp dissection with microscissors around the aneurysm neck and dome is useful for better outlining the aneurysm structure and decreases the risk of intraoperative rupture. Perforators should be identified, dissected, and preserved before clip placement.

20.3.3 Temporary Clip Placement

Temporary clip placement softens the aneurysm and allows for the final stages of aneurysm dissection, allows the neurosurgeon to manipulate the aneurysm, decreases the risk of intraoperative aneurysm rupture, and facilitates permanent clip placement on the aneurysm neck. The authors do this under barbiturate electroencephalographic burst suppression for cerebral protection. Usually, one temporary clip is sufficient, placed on the afferent artery, and trapping by temporary clipping of efferent branches can be used if needed.

20.3.4 Clipping Strategy

The efficacy of microsurgical clipping in the setting of a prior endovascular intervention depends on several factors. Prior to clip placement, the dimensions of the aneurysm should be carefully outlined, and the position of the coils within the aneurysm, the presence of coils extrusion, and the space available at the base of the aneurysm for clip application should be recognized. Estimating the length of the neck of the aneurysm remnant or recurrence after previous endovascular treatment is of utmost importance. As with clipping of any aneurysm, an adequate neck is required for proper clip placement to reduce the risk of damage to the parent artery. With previous endovascular interventions, coils may be found within the aneurysm neck, reducing the available space for clip placement. Therefore, microsurgical clipping would be most effective when the height of the aneurysmal remnant is at least twice that of the neck. A single clip placed at the base of the aneurysm neck will result in successful occlusion of the aneurysm and preservation of the parent artery. When the neck of the aneurysm is too short, the risk of injury to the parent artery is augmented and, in this case, reconstructive multiple clip placement is the method of choice.

The removal of coils from the aneurysm is usually avoided prior to clip placement since it is associated with significant morbidity and worse outcomes. This is especially true when the time interval between the endovascular procedure and the clipping procedure is longer. A longer time will result in significant scarring and thinning of the aneurysm wall, making the process of coil extraction harder. Nevertheless, difficulties attributed to coils present within the aneurysm neck are frequently seen and sometimes proper clip application necessitates the removal of part of these coils. When the coils are densely scarred into the aneurysm or when the coils extrude into the subarachnoid space, coil extraction may be necessary for clip closure. Coil removal should be performed with temporary trapping of the aneurysm. We usually do this under barbiturate burst suppression with modest hypothermia to lower cerebral metabolic requirements and improve cerebral protection. Inspection for and removal of any coils or thrombus fragments in the lumen of the parent artery should be performed before final clipping is performed to avoid the occurrence of thromboembolic complications. The perforating vessels adjacent to the aneurysm are sometimes found to be densely adhering to the aneurysm or extruded coil mass and should be carefully dissected to preserve them.

Although the main surgical treatment option for previously endovascularly treated aneurysms is direct microsurgical clipping, other methods may be necessary in certain situations. Prior coil placement can result in significant scarring, calcification, and thrombosis, affecting the aneurysm wall, and the coils may extrude into the aneurysm neck. Clip application in these more difficult cases may be unsafe. This may entail the performance of aneurysm wrapping, bypass and trapping, or proximal vessel occlusion.

Recurrence of stented aneurysms, although less frequent, is even more challenging to manage with microsurgical clipping. A stent in the parent artery, especially when it has been placed for a long time, adheres to the parent vessel wall, making it rigid, noncompliant, and difficult to manipulate. Furthermore, the application of a temporary clip can result in modification of the previously placed stent. The removal of the stent is not recommended. Clip placement close to the parent artery can result in tearing of the aneurysm or the parent vessel at its base. The clips should be placed to allow just enough room in the neck for the clips to close. Aneurysms that have been previously coiled and stented are even more challenging to manage surgically because of the rigidity of the structures and the little space available to perform necessary maneuvers.

20.3.5 Intraoperative Angiography

After clipping of the aneurysm, micro-Doppler recordings are useful in assessing the patency of the afferent and efferent vasculature and to check for residual flow within the aneurysm. Intraoperative angiography is useful to document aneurysm occlusion and patency of the afferent and efferent arteries. This is especially useful when clipping is performed after a prior endovascular intervention because of the heightened uncertainties that accompany the procedure. Furthermore, if a bypass procedure was performed, intraoperative angiography should be used to evaluate the patency of the anastomosis. To perform intraoperative angiography, bilateral groins should be prepared and draped in a standard sterile fashion, and then a femoral artery introducer sheath should be inserted into the femoral artery opposite to the craniotomy site via the Seldinger technique and connected to a heparinized saline flush. It is best to obtain femoral access after induction of anesthesia and before surgery. Gaining femoral access at this point avoids the need for radial arterial line placement and minimizes the time needed to obtain an angiogram which will allow for a timely diagnosis and management if an unsuspected arterial occlusion is identified. It also avoids a hurried and stressful struggle in the event of suboptimal clip application when the surgeon is worried about but unsure of parent artery flow. Intraoperative angiography studies can be acquired under a portable single-plane fluoroscopy unit. Angiograms are then immediately reviewed by the neurosurgeon for evidence of residual pathology and patency of parent/branch vessels. This allows for prompt intraoperative adjustments. Intraoperative angiography is safe and carries a minimal risk of morbidity (< 1%). Indocyanine green fluorescence angiography is another intraoperative tool that can provide a rapid evaluation of the vessel patency, aneurysm occlusion, and anastomotic success.

20.4 Postoperative Management Including Possible Complications

Any new changes or deficits during or after the procedure can indicate the presence of vessel compromise or thromboembolic or hemorrhagic complications and should be detected as soon as possible. The information obtained from neurophysiologic monitoring should be assessed during and after clip placement and a neurologic exam should be obtained immediately following the procedure to identify any new deficits. Integration of the electrophysiological and clinical findings allows for prompt assessment and management of postoperative complications. If there is suspicion of compromise to the patency of the parent artery, injury to the perforator vessels, or improper clip placement, immediate re-exploration should be considered. A noncontrast head CT scan should be obtained if the patient awakens with a focal neurologic deficit or if the patient's awakening is delayed in order to rule out intracranial hemorrhage (to note that sometimes the patient might not awaken for hours after burst suppression). If the deficits cannot be explained by clinical, neurophysiologic, and radiologic data, cerebral angiography may help elucidate the cause.

Patients who underwent microsurgical management of previously endovascularly treated aneurysms should be transferred to the neurological intensive care unit postoperatively for monitoring of cardiopulmonary and hemodynamic status, blood volume, and blood pressure. Prophylaxis against deep vein thrombosis may be initiated. We use intermittent pneumatic compression stocking and we generally start heparin (5,000 units) subcutaneously every 12 hours starting 12 hours postoperatively. Gastric ulcer prophylaxis and seizure prophylaxis should be considered as well. Prolonged use of invasive vascular lines and indwelling catheters should be avoided to minimize the risk of infection.

With the growing population of aneurysm recurrences and residuals following endovascular management, treatment guidelines should be established to identify the best management course for these aneurysms. Surgical clipping is an appropriate treatment of recurrent and residual cerebral aneurysms that failed endovascular management or when endovascular intervention is no longer an option. Although clipping of previously endovascularly treated aneurysms carries higher operative risks than microsurgical management of naïve aneurysms, a high success rate (94–100%),[5,6,7] acceptable complication rate (around 10%),[1,8] and a low retreatment rate can be achieved. Direct clipping of the aneurysm neck can be performed in most patients; however, if there is little space between the coil mass and the parent vessel, a bypass procedure should planned preoperatively in case clip ligation is not feasible. Coil extraction should not be routinely performed because of increased morbidity. Retreatment of previously endovascularly treated aneurysms can be performed using another endovascular approach or surgical management. Management should be individualized based on patient characteristics, remnant/recurrence anatomy, and the neurosurgeon's expertise.

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21 Pterional Craniotomy for Exposure of Contralateral Aneurysms

Joao Paulo Almeida, Danilo Silva, Judy Huang, and Rafael J. Tamargo

Abstract

Between 14 and 34% of patients with intracranial aneurysms have more than one aneurysm. When feasible, the ability to treat these patients through a single, unilateral, craniotomy offers the advantages of avoiding the risk of multiple craniotomies and of decreasing hospital length of stay and costs. Clipping multiple ipsilateral aneurysms is well described and is also possible for bilateral midline aneurysms typically at the anterior communicating or distal anterior cerebral arteries. This chapter discusses the indications, surgical techniques, and clinical results of intracranial aneurysms treated through a contralateral approach. Understanding the microsurgical nuances of the basal cisterns and anterior circulation anatomy is required for successful contralateral approaches. The natural corridors provided by the cisterns provide space for surgical navigation toward the contralateral side, without the need of transgression of brain parenchyma. Through adequate surgical dissection and use of the cisternal spaces, candidates for contralateral approaches include ophthalmic, carotid bifurcation, posterior communicating segment, and proximal middle cerebral artery aneurysms.

Keywords: intracranial aneurysm, craniotomy, multiple aneurysms

21.1 Introduction

Approximately 14 to 34% of patients with intracranial aneurysms have more than one aneurysm.[1] These multiple aneurysms are bilateral in 20 to 40% of cases.[1,2] When feasible, the ability to treat these patients through a single, unilateral, craniotomy offers the advantages of avoiding the risk of multiple craniotomies and of decreasing hospital length of stay and costs.[3,4,5]

Bilateral anterior circulation aneurysms, including ophthalmic segment, middle cerebral artery (MCA), posterior communicating segment, and internal carotid artery (ICA) bifurcation aneurysms, may be submitted to contralateral clipping. Adequate patient selection and surgical planning according to anatomical variations and location of the lesion are required for success of the procedure. Cases appropriate for contralateral approaches are infrequent and amount to approximately 10% of cases in the author's experience. Successful contralateral clipping is usually accomplished in about 80% of unruptured cases but in only about 40% of ruptured cases.[5,6]

21.2 Patient Selection

21.2.1 Multiple Intracranial Aneurysms

The majority of patients with multiple aneurysms harbor 2 aneurysms, although as many as 13 aneurysms have been described in a single patient. Approximately half of multiple aneurysms occur on different sides.[1,6] Bilateral carotid-ophthalmic segment aneurysms, usually in females, are one of the most common situations of multiple aneurysms.

In appropriately selected patients for surgical treatment of intracranial aneurysms, a common objective for treatment of multiple supratentorial aneurysms is to treat the ruptured or symptomatic aneurysm first via an ipsilateral pterional craniotomy as well as other ipsilateral aneurysms, except in cases of ruptured aneurysms where exposure of additional aneurysms would be difficult. Typically, one returns at a subsequent time to clip the any remaining contralateral aneurysms via a second pterional craniotomy. This is based on the central tenet of intracranial aneurysm surgery of obtaining the requisite proximal vessel control during microdissection.

Using the contralateral approach, aneurysms of the precommunicating segment of the anterior cerebral artery and the ICA bifurcation are observed in a straight line and can be clipped with relative ease with minimal risk of injury to the perforating arteries. Aneurysms arising from the posterior communicating or anterior choroidal arteries point posterior and lateral, making repair from the contralateral side difficult. MCA aneurysms, particularly when the M1 segment is short, can also be explored from the contralateral side.[7] Avoidance of a separate, second craniotomy to treat contralateral, incidentally detected aneurysms is desirable because it avoids a waiting period for treatment of a known unruptured aneurysm, as well as additive risks, recovery time and costs of a separate craniotomy or embolization procedure, general anesthesia, and hospital stay.[3,4,8] Disadvantages of approaching unruptured aneurysms from the contralateral side include increased depth of exposure, lack of easily accessible proximal control, and need for additional dissection and brain retraction in some cases. Lack of proximal control, exposure, and brain retraction are issues in the setting of acute hemorrhage when the brain is swollen and anatomy is obscured by blood.

Currently, the other option for incidental aneurysms with appropriate anatomical characteristics is endovascular coil embolization instead of a second craniotomy. It is important to consider the development of endovascular techniques as an additional tool, especially for those who present with subarachnoid hemorrhage and contralateral aneurysms. Endovascular coiling may be chosen whenever a contralateral approach seems too risky.

21.2.2 Carotid-Ophthalmic Segment Aneurysms

Carotid-ophthalmic segment aneurysms frequently arise from the medial wall of the ICA and are often bilateral. This renders a subset of cases of nongiant aneurysms to be particularly well suited for a contralateral approach (▶ Fig. 21.1). Optimal results from microsurgical treatment of carotid-ophthalmic segment aneurysms necessitate understanding of the anatomical relationship of these aneurysms to the distal dural ring at the roof of the cavernous sinus, anterior clinoid process including the optic strut, falciform ligament, optic nerve, and chiasm, origins of the ophthalmic and superior hypophyseal arteries, and pituitary stalk.[6,7] The limited working space of the paraclinoidal area

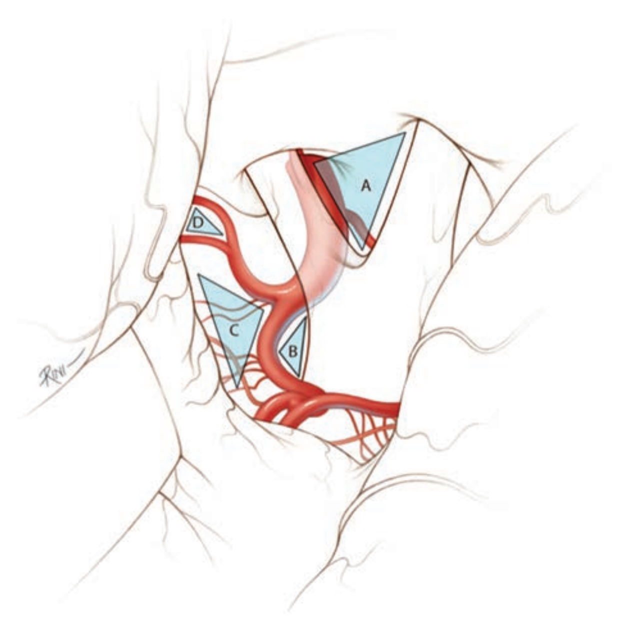

Fig. 21.1 Exposure of contralateral aneurysms at the: *A*, ophthalmic region; *B*, posterior communicating segment; *C*, carotid termination; and *D*, middle cerebral artery, and their relationships to the triangular subarachnoid spaces.

makes avoidance of trauma to the optic nerve and chiasm during microdissection of paramount importance. Complete division of the falciform fold is essential to permit safe mobilization of the optic nerve. Intradural drilling is usually unnecessary for small aneurysms, and if partial removal of the tuberculum sellae is required, this is more easily accomplished than the removal of the anterior clinoid process and the optic strut that is necessary in ipsilateral approaches.[5] Size, location, morphology, and dome projection of carotid-ophthalmic aneurysms should be carefully evaluated for optimal patient selection.[9] Saccular aneurysms, smaller than 10 to 15 mm, that displace the optic nerve superolaterally and the optic chiasm superiorly, arising from the medial or superomedial wall of the ICA, are suitable candidates to be approached from a contralateral approach (▶ Table 21.1). Complex aneurysms (giant, fusiform, bilobulate, the presence of wall irregularities and calcifications) are preferentially approached via an ipsilateral craniotomy.[5,6,7]

Since the most common route for contralateral clipping of those aneurysms is through the interoptic space, patients who present with short interoptic space and/or a prefixed chiasm may not be ideal candidates for this approach. Preoperative evaluation with 3D reconstruction computed tomography scans and analysis of the distance between the optic nerves as well as distance between the ICAs at the level of the tuberculum may be helpful.[9,10]

Relative contraindications to the contralateral approach include a prefixed optic chiasm, giant aneurysms, large ruptured aneurysms, and aneurysms that originate laterally or displace the optic nerve superomedially.

21.3 Operative Procedure

The exposure of contralateral aneurysms begins with the patient positioned for a standard pterional craniotomy. To minimize brain retraction, maximize microscopic illumination for anatomical visualization, and enlarge the space for working corridors to the contralateral vessels, meticulous extension of the fronto-orbital exposure is recommended. This involves removal of the inner table of the medial and inferior frontal bone at the anteromedial extent of the craniotomy and flattening of the irregular surface of the ipsilateral orbital roof and sphenoid wing with a high-speed drill. The greater and lesser medial sphenoid wings adjacent to the sylvian fissure are drilled flat to connect the lateral extent of the anterior cranial fossa with the anterior extent of the temporal fossa.[5,6]

After the dura is opened and tented flat against the sphenoid wing, the microscope is brought in to the field and sharp arachnoid microdissection is initiated with entry into the sylvian cistern at the region of the frontal operculum. Wide opening of the sylvian fissure diminishes the need for frontal lobe retraction and provides a broad corridor for dissection of the basal cisterns. Arachnoid dissection continues into the ipsilateral opticocarotid cistern. Complete exposure of the optic apparatus is necessary for orientation. The release of cerebrospinal fluid (CSF) from the basal cisterns will allow brain relaxation. In cases of thick subarachnoid clot in the basal cisterns in which brain relaxation is anticipated to be inadequate via CSF release from opening of the basal cisterns, a lumbar subarachnoid catheter or intraventricular catheter can be used. After the ipsilateral optic nerve is released from the frontal lobe, the chiasmatic cistern is opened. Further brain relaxation may be accomplished by release of CSF from the interpeduncular cistern with opening of the membrane of Liliequist and opening of the ventricular system by fenestration of the lamina terminalis (▶ Fig. 21.2). Continued contralateral dissection then begins with opening of the contralateral optic and carotid cisterns (▶ Fig. 21.3).

Table 21.1 Correlation between triangular spaces and aneurysm subtype approached by a contralateral pterional craniotomy[3]

Triangular space	Aneurysm type
The interoptic space	Carotid-ophthalmic and superior hypophyseal artery aneurysms projecting medially Posterior communicating and anterior choroidal segment aneurysms projecting inferiorly
The opticocarotid space	Posterior communicating and anterior choroidal artery aneurysms projecting posterolaterally
The supracarotid space	Internal carotid artery bifurcation aneurysms projecting superiorly
Middle cerebral artery space	Middle cerebral artery bifurcation aneurysms projecting superiorly or inferiorly

21.3.1 Contralateral Exposure of Carotid-Ophthalmic Segment Aneurysms

Complete exposure of the optic chiasm is followed by dissection of the interoptic space. This triangular space that is bordered by the planum sphenoidale anteriorly and optic nerves bilaterally is dissected to gain access to the contralateral ophthalmic artery origin and superior hypophyseal artery origins that are medial. Prior to dissection of the medial wall of the contralateral carotid

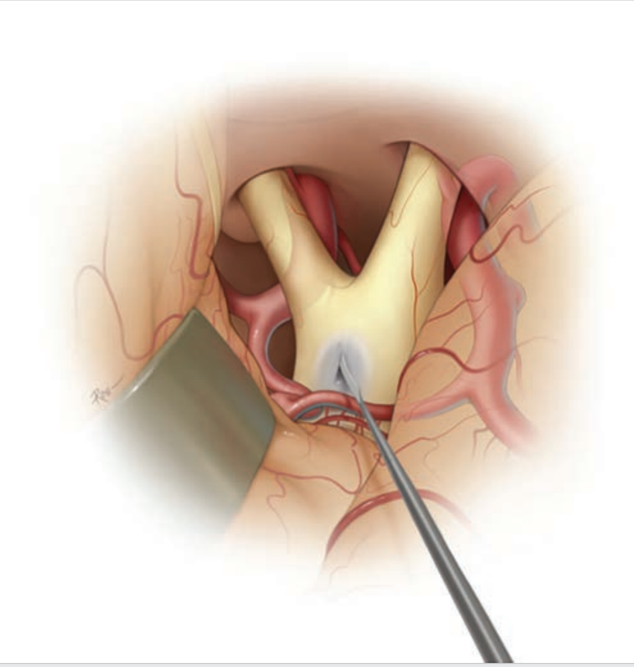

Fig. 21.2 Opening of the lamina terminalis with use of a sickle knife or microsurgical bipolar allows further drainage of CSF, reduces the need of retraction over the brain parenchyma, and improves the exposure of contralateral vascular structures.

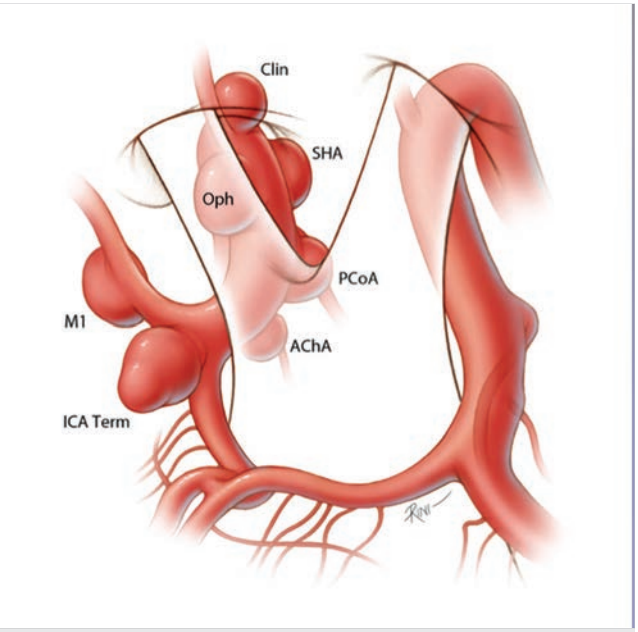

Fig. 21.3 Exposure of the contralateral carotid bifurcation, A1, and M1 branches, after extensive opening of the basal cisterns and lamina terminalis.

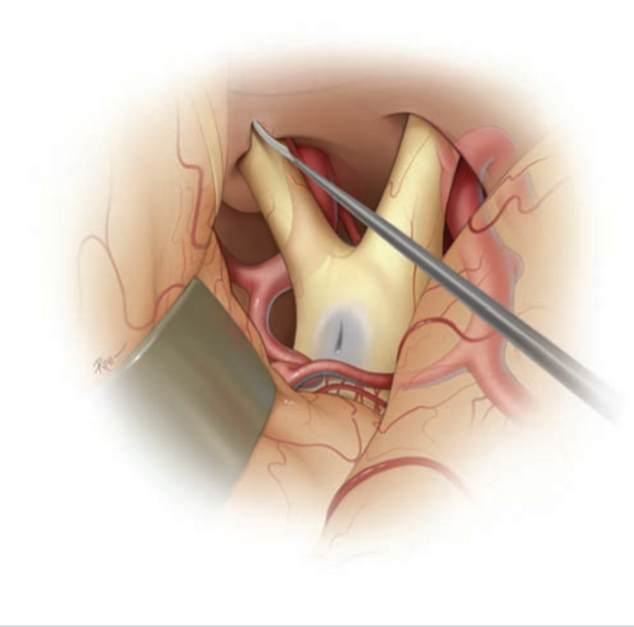

Fig. 21.4 Opening of the contralateral falciform ligament is carefully performed with a sickle knife in order to achieve further mobilization of the contralateral optic nerve and exposure of ophthalmic and superior hypophyseal aneurysms.

artery from which the contralateral ophthalmic segment aneurysm arises, mobilization of the contralateral optic nerve is necessary to diminish traumatic nerve injury during manipulation. This is accomplished by longitudinal incision of the contralateral falciform ligament at the center of its circumference as far as the roof of the optic canal[6,7] (▶ Fig. 21.4). Rarely, further untethering of the contralateral optic nerve is possible with unroofing of the contralateral optic canal with a high-speed drill and small diamond burr. Generous irrigation is performed while drilling close to the optic nerve in order to avoid thermal injury of the nerve. After such maneuvers, mobilization of the contralateral optic nerve laterally enables visualization of the medial wall of the paraclinoid ICA. It is through this interoptic space that the neck of small carotid-ophthalmic and superior hypophyseal artery aneurysms that project medially may be defined for suitable clip placement (▶ Fig. 21.5 and ▶ Fig. 21.6).

Options for proximal vascular control include direct clip occlusion of the paraclinoid ICA proximal to the origin of the ophthalmic artery, neck dissection to access the cervical ICA, or endovascular transfemoral balloon occlusion of the petrous segment of the ICA. The method selected depends on the aneurysm size, anticipated complexity of aneurysm dissection, and institutional preference. Vascular control proximal to the origin of the aneurysm may not be feasible without opening of the distal dural ring, a difficult maneuver to be performed through the narrow corridor of a contralateral approach. Therefore, ophthalmic aneurysms arising from short paraclinoid ICAs may require neck dissection for adequate proximal control.

A prefixed optic chiasm resulting in a limited interoptic space or aneurysms that project laterally or superolaterally may hamper this contralateral approach to ophthalmic segment aneurysms. An ipsilateral approach to large and giant ophthalmic aneurysms remains preferred due to the need for adequate removal of the anterior clinoid process and optic strut in these situations.[9]

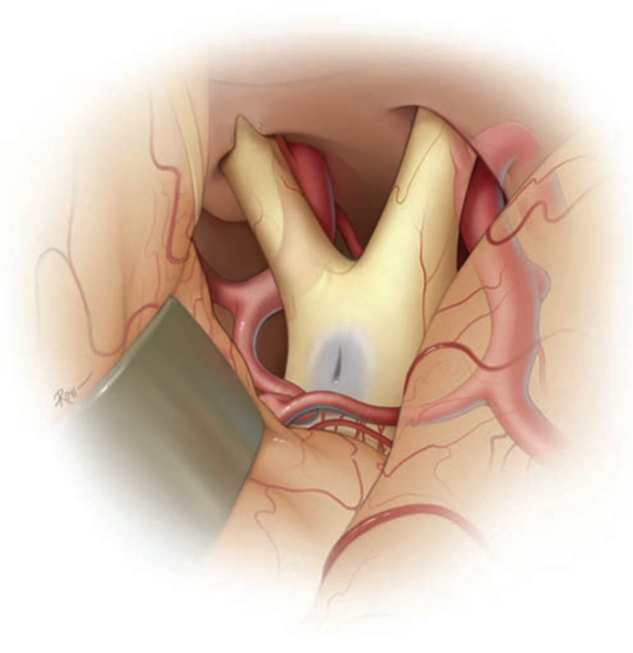

Fig. 21.5 Improved exposure of the medial wall of the contralateral paraclinoid ICA after opening of the falciform ligament.

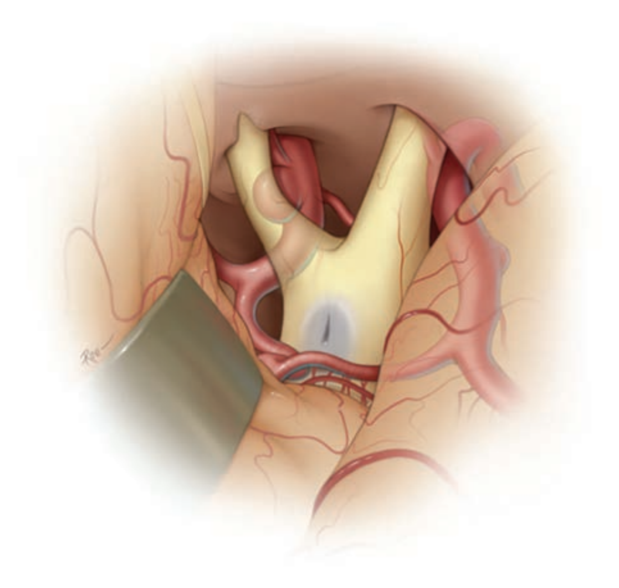

Fig. 21.6 Exposure of contralateral ophthalmic aneurysm after extensive opening of the basal cisterns.

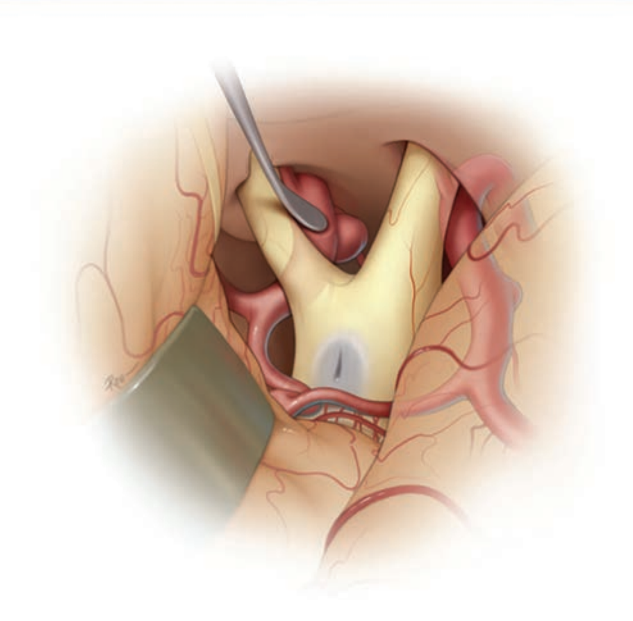

Fig. 21.7 Contralateral posterior communicating artery aneurysm. Exposure and clipping of those lesions is usually achieved after dissection of the interoptic space. Medially projecting aneurysm may be suited for this approach; however, aneurysms that arise from the lateral wall of the internal carotid artery (ICA) at this region are poor candidates for contralateral clipping.

21.3.2 Contralateral Exposure of Posterior Communicating and Choroidal Segment Aneurysms

Exposure of contralateral posterior communicating and choroidal segment aneurysms may be accomplished either through the interoptic space or through the contralateral opticocarotid space (▶ Fig. 21.1, ▶ Fig. 21.2, ▶ Fig. 21.3, ▶ Fig. 21.4, ▶ Fig. 21.5, ▶ Fig. 21.6, ▶ Fig. 21.7, ▶ Fig. 21.8). Aneurysms at these locations are the most difficult aneurysms to expose contralaterally as they are partially hidden by the carotid artery and optic nerve. Boundaries of the triangular contralateral opticocarotid space are: the lateral aspect of the contralateral optic nerve, chiasm, and tract; the medial aspect of the contralateral ICA; and the inferior aspect of the precommunicating segment of the contralateral anterior cerebral artery. Through this space and superior to the optic apparatus, the contralateral ICA may be mobilized laterally or medially to define the neck of posterior communicating and anterior choroidal artery aneurysms.[6] Because these aneurysms frequently project posterolaterally, the neck region and origins of the posterior communicating and anterior choroidal arteries may be partially obscured by the contralateral ICA and optic apparatus. Adequate visualization depends on the relative position of the optic chiasm. Exposure is aided by the presence of a long contralateral supraclinoid carotid artery and a relatively distal origin of the contralateral posterior communicating artery. Prefixed chiasms can make the exposure either very difficult or even impossible. Clipping of

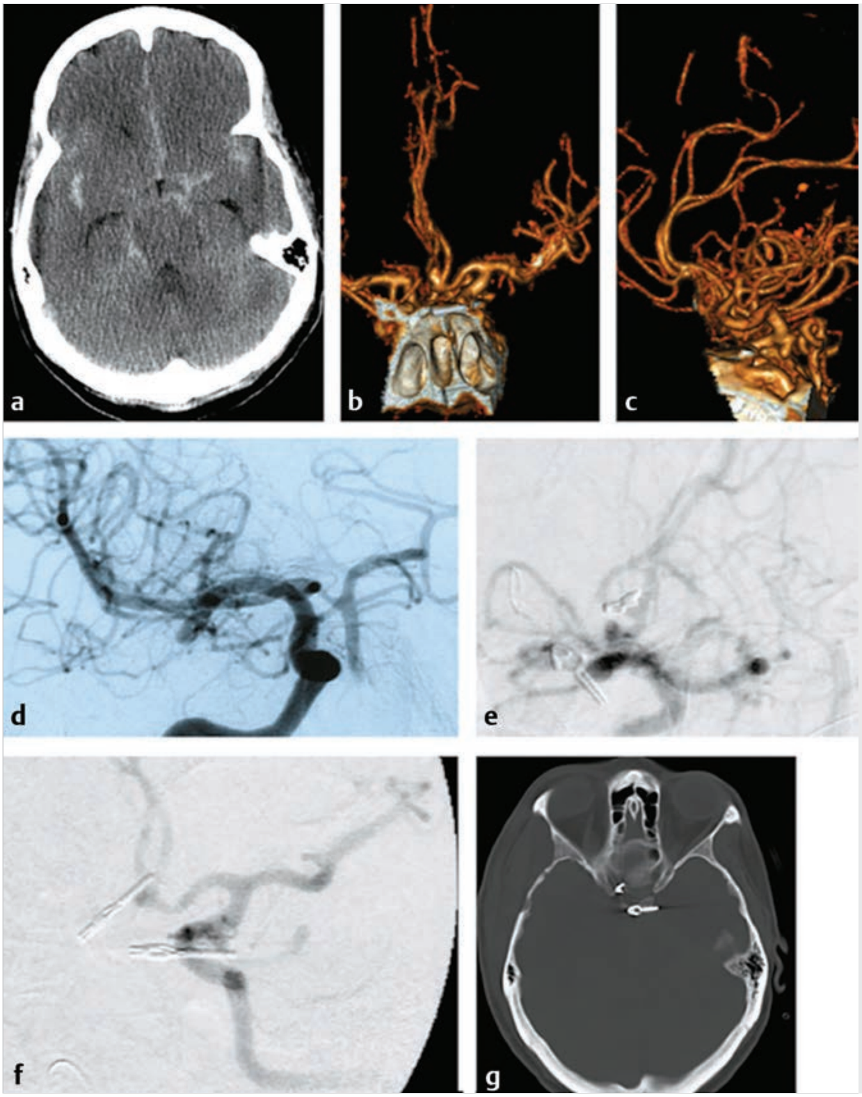

Fig. 21.8 Representative case of contralateral clipping of a posterior communicating artery aneurysm. A 55-year-old woman presented with subarachnoid hemorrhage (SAH), World Federation of Neurological Surgeons grade 2, and it was decided to treat all aneurysms through a single, supratentorial craniotomy. **(a)** The admission computed tomography (CT) scan showed diffuse, relatively symmetric SAH, and CT and catheter angiography showed aneurysms at **(b)** the anterior communicating artery (anteroposterior CT angiogram), **(c)** left posterior communicating artery (lateral CT angiogram), and **(d)** right middle cerebral artery (anteroposterior right internal carotid angiogram). All aneurysms were clipped through a right craniotomy. **(e)** Intraoperative left internal carotid oblique and **(f)** lateral carotid angiograms show adequate clipping of the anterior and posterior communicating aneurysms and patency of the posterior communicating artery. **(g)** The postoperative CT scan shows the clip on the posterior communicating artery coming in from the right side. (The images are provided courtesy of R. L. Macdonald, MD.)

aneurysms that project posteriorly will depend fundamentally on the position of the optic chiasm. When clipping is feasible, it can be done either above or below the chiasm.[7]

21.3.3 Contralateral Exposure of Internal Carotid Artery Termination Aneurysms

Aneurysms located at the contralateral internal carotid termination are best suited to a contralateral exposure. Those aneurysms are usually difficult to manage when approached from the ipsilateral side. The longer the carotid artery is, the greater the necessity for retraction of the frontal lobe. Retraction can cause aneurysm rupture in cases where the dome of the aneurysm is either located inside or fixed to the anterior perforated substance. The perforating arteries from the choroidal segment of the ICA are very difficult to identify, as the aneurysm walls usually cover them. Usually, the exposure of such aneurysms requires that the operative microscope be moved more laterally to avoid strong retraction of the frontal lobe. Placement of the aneurysm clip parallel to the A1 and M1 segment is seldom possible. In cases of aneurysms with broad necks, there is a tendency for the clip to cause deformities of the carotid bifurcation and subsequent blood flow problems.[7]

When a contralateral approach is selected, clipping of these lesions may be relatively straightforward. This is because the aneurysm will project upward toward the anterior perforated substance in a plane that is perpendicular to the working corridor and line of sight (▶ Fig. 21.9). The carotid termination is reached by following the contralateral A1 segment of the anterior cerebral artery proximally or the contralateral ICA distally to the carotid bifurcation. Further dissection distally on the first segment of the contralateral MCA and elevation of the contralateral frontal lobe reveals the contralateral supracarotid space. The boundaries of this triangular space are the contralateral sphenoidal segment of the MCA, precommunicating segments of the anterior cerebral arteries, and the contralateral basomedial frontal lobe. Even large carotid termination aneurysms are readily exposed through this space.[6] The perforating vessels that originate from the choroidal segment are dissected free from the posterior wall of the aneurysm, and the origins of the anterior cerebral artery and MCA are visualized. Clip placement may be accomplished parallel to the axis of both anterior

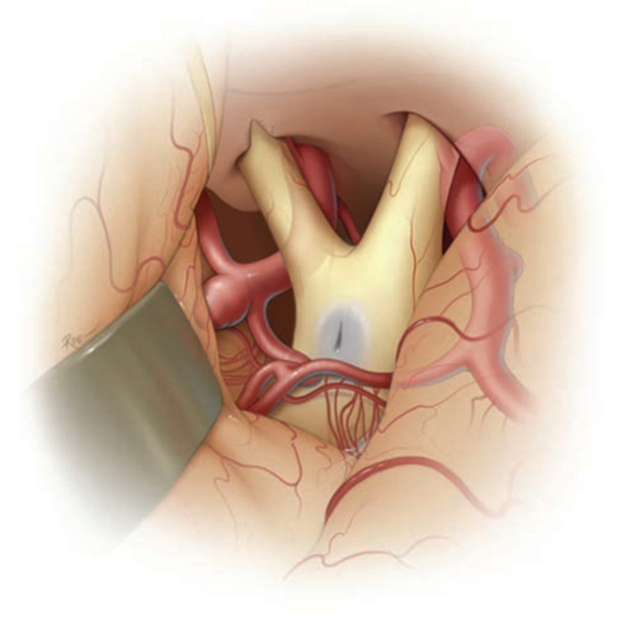

Fig. 21.9 Contralateral carotid bifurcation aneurysms. Those are some best lesions for contralateral clipping. Their upward projection toward the anterior perforated substance facilitates exposure of the neck of the aneurysm and clipping. Perforating branches are usually around the dome of the aneurysm and careful dissection must be performed prior to final clipping of the lesion.

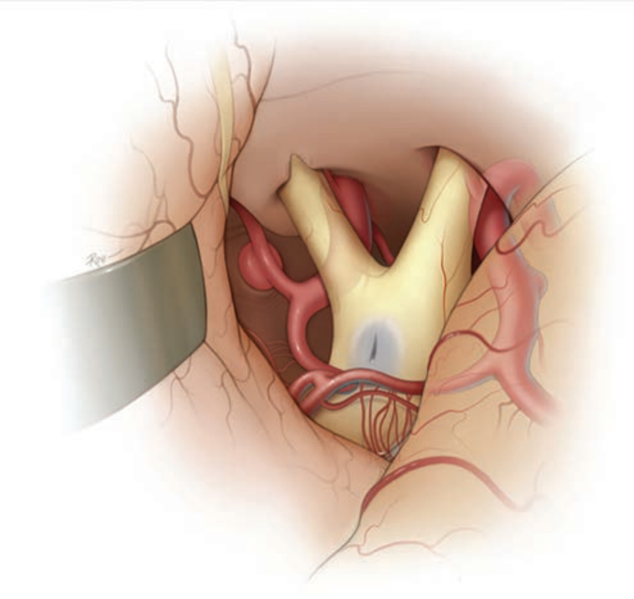

Fig. 21.10 Contralateral middle cerebral artery (MCA) aneurysms. Clipping of contralateral MCA aneurysms may be performed in lesions located proximal to the limen insula. Extensive opening of the basal cisterns and lamina terminalis and identification and preservation of lateral lenticulostriate arteries is performed for successful treatment of those aneurysms.

cerebral artery and MCA segments with less risk of perforating artery compromise than that associated with an ipsilateral approach.

21.3.4 Contralateral Exposure of Middle Cerebral Artery Bifurcation Aneurysms

After the carotid termination is reached and the supracarotid space opened, the contralateral sphenoidal segment of the MCA is followed distally to the bifurcation by dissection along its inferior surface. Adherence to the inferior surface of the MCA during sphenoidal segment dissection avoids injury to the lateral lenticulostriate arteries, which typically arise from the superior surface of the vessel toward the anterior perforated substance. Exposure of contralateral MCA bifurcation aneurysms is possible up to the level of the contralateral limen insula (▶ Fig. 21.10). MCA aneurysms distal to the limen insula are not suitable for contralateral clipping.

When dealing with bilateral MCA aneurysms, clipping of the ipsilateral lesion may be postponed until the contralateral aneurysm is treated in order to avoid view obstruction by surgical clips.[8]

Ease of access to these aneurysms is facilitated by the presence of a relatively short sphenoidal MCA segment. MCA bifurcation aneurysms that project anteriorly or inferiorly toward the sphenoid wing are more amenable to clip obliteration because they are perpendicular to the line of sight and may be clipped with a simple straight clip. Laterally projecting MCA bifurcation aneurysms are more challenging to treat from a contralateral exposure given that the parent artery may obscure visualization during isolation of the aneurysm neck. Parent artery and aneurysm mobilization may be required for adequate exposure of the lesion, which is facilitated with temporarily clipping of the M1 segment and softening of the aneurysm. Laterally projecting aneurysms are clipped with a curved clip, with the tips curving downward across the neck. A second curved clip, with the tips curving upward across the neck and intersecting the blades of the first clip, may be needed when the first clip does not completely occlude the neck.[8] Superiorly projecting aneurysms may hide behind the medial orbital gyrus and are exposed by mobilizing the aneurysm inferiorly, retracting the frontal lobe superiorly, or resecting a small portion of overlying gyrus (▶ Fig. 21.4). Once visualized, the superior projection of these aneurysms makes them easy to clip with a straight clip.

21.4 Clinical Results

In the late 1970s, the pterional craniotomy was first reported by Yasargil et al as a useful approach to clip contralateral aneurysm.[11] They described five cases of bilateral carotid-ophthalmic aneurysms successfully clipped through a unilateral pterional craniotomy, and pointed out that clipping of the contralateral aneurysm was feasible "whether the fundus of the aneurysm is directed medially or laterally." Despite the development of endovascular therapies for the treatment of intracranial aneurysms during the last two decades, the contralateral approach for aneurysm clipping is still useful for treatment of selected cases. Encouraging clinical results achieved by this approach have been reported by multiple authors.[2,5,7,8,9,10]

Carotid-ophthalmic segment aneurysms were the first subtype of aneurysms treated via a contralateral pterional craniotomy.[11] Fries et al published a series of 51 patients with 58 carotid-ophthalmic aneurysms in which 9 patients with 10 aneurysms were treated through a contralateral pterional craniotomy.[12] Nineteen patients presented with uni- or bilateral visual disturbances. Three patients experienced visual deterioration after surgery, 1 patient remained stable, and 15 of them experienced improvement in visual function. The patients who presented with visual deficits and were submitted to a contralateral approach experienced visual improvement. All the patients whose visual function deteriorated after surgery were approached via the unilateral craniotomy. The authors suggested that manipulation of the optic structures was responsible for worsening visual function when the aneurysms were clipped using a unilateral approach. In one of the largest series of carotid-ophthalmic aneurysms approached by a contralateral craniotomy, Andrade-Barazarte et al[9] reported optimal clinical results with 28 of 30 patients presenting at 3-month follow-up visit with a modified Rankin Scale (mRS) score of 0 to 3 and only 1 patient experiencing postoperative visual deficit described as hemianopia. Of the two patients who exhibited poor clinical outcome at 3 months, none of them was related to the approach itself. One patient developed vasospasm and the other presented with poor clinical grade preoperatively.

In an elegant descriptive anatomical study, de Oliveira et al[7] reported the results of 51 patients harboring 55 aneurysms approached by a contralateral craniotomy with no mortality or morbidity related to the contralateral route. The authors concluded that aneurysms most suited for a contralateral approach are the following: carotid-ophthalmic aneurysms, aneurysms of the posterior communicating and anterior choroidal segment of the ICA, and aneurysms located at the A1 and M1 segment of the anterior cerebral artery and MCA, respectively. Lynch and Andrade reported “excellent” or “good” outcome (independent life-style and employment) in 13 out of 15 patients in whom the authors were able to clip 15 contralateral aneurysms through a unilateral pterional craniotomy.[2] Using an extended unilateral orbitopterional craniotomy, Rajesh et al were able to treat 10 patients harboring 23 aneurysms, 12 located on the contralateral side, achieving good clinical results with Glasgow Outcome Scale (GOS) IV or V in 9 of them.[13] In a series of 46 patients with bilateral MCA aneurysm published by de Sousa et al using the unilateral pterional approach, complete obliteration by surgical clipping was obtained in 30 patients (65%).[14] The vast majority of patients experienced optimal clinical outcome, with 90% of patients presenting at last follow-up with GOS IV or V. The main reasons for failure of clipping the contralateral MCA aneurysm stated by the authors were the presence of brain edema, in the acute setting of hemorrhage, lateral projection of the aneurysm, and very long M1 segment.[14] A comparative study by Rodríguez-Hernández et al,[8] which evaluated unilateral and bilateral approaches to bilateral MCA aneurysms, confirmed the safety, clinical efficacy, and cost efficiency of unilateral approach to bilateral MCA aneurysms. Good neurological outcome, described by the mRS score of 0 to 2, was equivalent in both groups, and the unilateral craniotomy group averaged roughly $20,000 dollars less per patient compared to the bilateral craniotomy group.[8] The authors concluded that this technique is best applied in older patients with sylvian fissures widened by brain atrophy and, also, in the setting of unruptured aneurysms pointing inferiorly or anteriorly with short M1 segments.

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22 Infectious Intracranial Aneurysms

Michael K. Tso and R. Loch Macdonald

Abstract

The term *mycotic aneurysm* was first coined by William Osler in 1885 to describe the gross pathological appearance of this "fungating" aneurysm. However, the term persists despite the majority of mycotic aneurysms being caused by bacterial (and not fungal) invasion of the blood vessel wall and subsequent dilatation. Hence, the preferred term is *infectious aneurysm*. Infectious intracranial aneurysms are relatively uncommon, representing approximately 2% of all intracranial aneurysms in published case series. However, the true incidence is unclear, especially due to the fact that infectious aneurysms can remain clinically silent and may regress in response to antibiotics without being detected. The wall of infectious aneurysms can be extremely friable. The vast majority of infectious intracranial aneurysms are cerebral arteries distal to the circle of Willis and are treated via endovascular means in modern published case series. However, surgical options still need to be considered. The interested reader is directed to the references for excellent comprehensive reviews on infectious intracranial aneurysms. This chapter provides an overview of the operative decision-making and management of infectious intracranial aneurysms, although specific surgical techniques are discussed elsewhere.

Keywords: infectious intracranial aneurysm, mycotic aneurysm, surgery, endovascular, subarachnoid hemorrhage; endocarditis

22.1 Patient Selection

22.1.1 Diagnosis

Clinical suspicion for an infectious intracranial aneurysm should be raised in a patient with bacterial endocarditis and acute onset of neurological symptoms.[1,2] The presumed pathophysiology of infectious aneurysm formation is hematogenous spread from bacteremia and/or septic emboli derived from valvular vegetations in the setting of bacterial endocarditis. Risk factors for developing endocarditis-related valvular vegetations include intravenous drug use, immunosuppression, and prosthetic arterial devices such as stents, grafts, and valves.[3] Contiguous spread from adjacent infection including orbital cellulitis with cavernous sinus thrombophlebitis, sinusitis, or meningitis may also lead to infectious aneurysm formation. There is a slight male predilection. Infectious aneurysms typically present in a ruptured state, with any combination of subarachnoid hemorrhage (SAH), intracerebral hemorrhage (ICH), intraventricular hemorrhage, and, rarely, subdural hemorrhage. The most frequent organisms isolated from blood cultures include *Streptococcus* species (including viridans), *Staphylococcus* species, and *Enterococcus* species. Fungal species are rare, and approximately one-third of cultures are negative. Unlike typical saccular aneurysms occurring at arterial bifurcation points around the circle of Willis, infectious aneurysms are frequently found in the distal middle cerebral artery (MCA) distribution and have a fusiform morphology.

A noncontrast computed tomography (CT) of the head will determine if there is a hemorrhage. Infectious aneurysms are typically diagnosed on a CT angiography (CTA). Magnetic resonance angiography (MRA) may also be used, especially in patients with poor renal function. However, time-of-flight MRA sequences may miss smaller, more distal aneurysms. Digital subtraction angiography (DSA) is the gold standard for detecting infectious aneurysms, especially very small distal aneurysms.

The differential diagnosis of a solitary spontaneous intraparenchymal hematoma should include a ruptured infectious aneurysm, particularly in the setting of endocarditis. Designating an intracranial aneurysm as an infectious aneurysm on imaging is based on the morphology (e.g., fusiform) and location of the aneurysm (e.g., distal MCA) in the appropriate clinical context (e.g., endocarditis or some other infection).

22.1.2 Indications for Surgery

The key to determining whether an infectious aneurysm requires surgical or endovascular treatment depends on the rupture status (▶ Fig. 22.1). Ruptured infectious aneurysms are thought to have a high risk of rebleeding and therefore tend to be treated aggressively. Unruptured infectious aneurysms may be followed with serial imaging during medical treatment of the infection, with surgery considered if the aneurysm grows, persists, or, obviously, ruptures. Patients with a life-threatening intraparenchymal hemorrhage are taken to surgery for evacuation of the hematoma and, if feasible, clip reconstruction or aneurysm trapping with or without bypass. Otherwise, endovascular therapy is often the first option since many of these patients are medically ill with severe cardiac disease and may be on antithrombotic drugs. Endovascular options include both direct coiling and embolization with liquid embolic agents (e.g., *n*-butyl-2-cyanoacrylate [NBCA] or ethylene vinyl alcohol copolymer dissolved in dimethyl sulfoxide mixed with tantalum powder [Onyx, ev3 Inc.]). Surgery is considered if the endovascular option is unfeasible or unsafe (e.g., unable to selectively catheterize the feeding artery or the parent artery supplies a potentially eloquent region). The goals of surgery are to prevent rupture or rebleeding.

22.1.3 Contraindications for Surgery

Surgery is not recommended in patients who are hemodynamically unstable or patients who are in a moribund state. Also, patients must have any coagulopathy corrected prior to surgery. Repair should be undertaken expeditiously in ruptured cases.

22.1.4 Timing of Surgery

Infectious aneurysms are typically pseudoaneurysms. As a result, the aneurysm wall may be quite fragile. Therefore, if endovascular options are not feasible, some surgeons recommend waiting several days to a week with the patient on antibiotics after aneurysmal rupture to allow for fibrosis, resulting in a

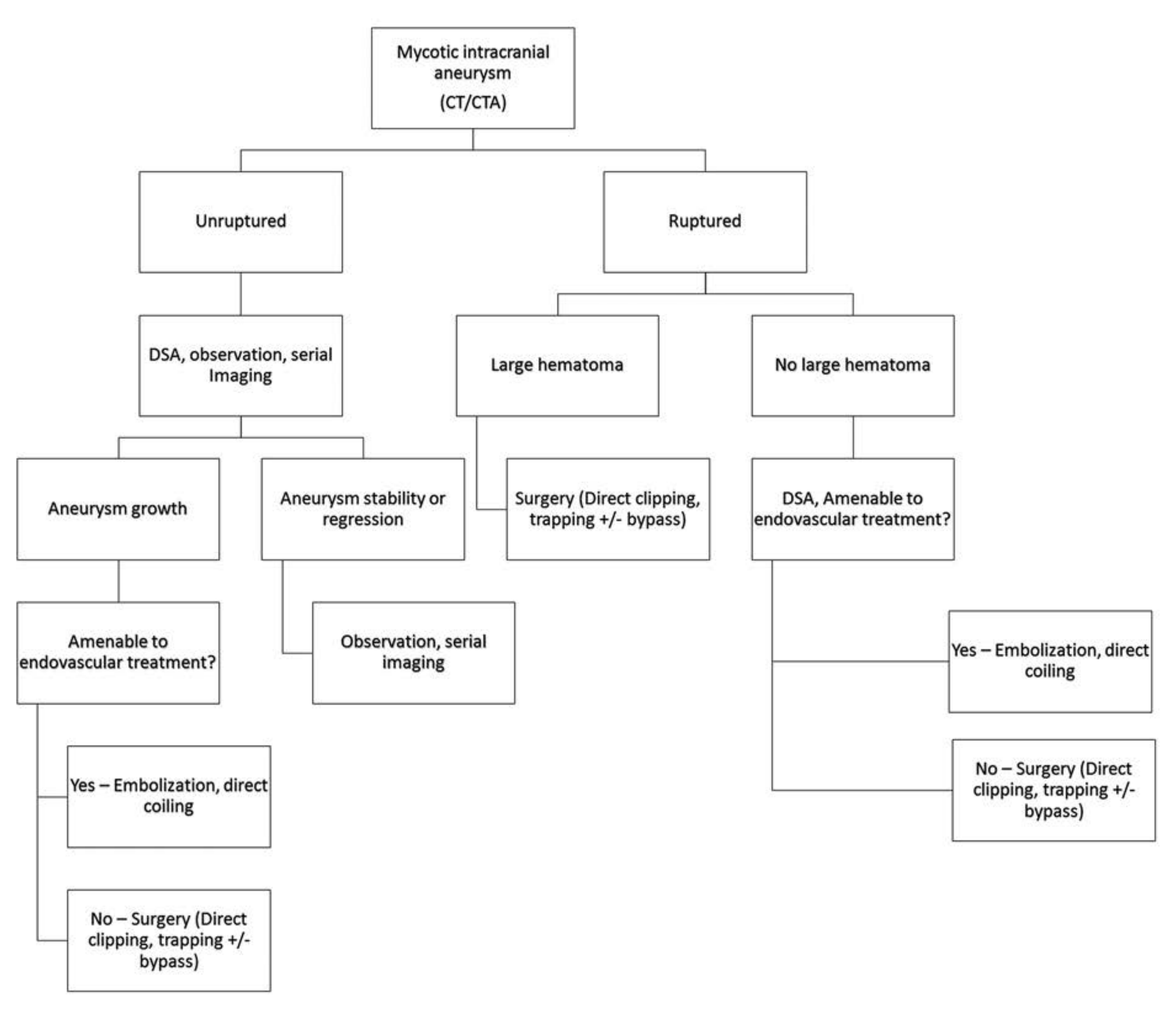
Mycotic intracranial aneurysm (CT/CTA)
Unruptured
Ruptured
DSA, observation, serial Imaging
Large hematoma
No large hematoma
Aneurysm growth
Aneurysm stability or regression
Surgery (Direct clipping, trapping +/- bypass)
DSA, Amenable to endovascular treatment?
Amenable to endovascular treatment?
Observation, serial imaging
Yes – Embolization, direct coiling
No – Surgery (Direct clipping, trapping +/- bypass)
Yes – Embolization, direct coiling
No – Surgery (Direct clipping, trapping +/- bypass)

Fig. 22.1 Management paradigm for patients presenting with suspected infectious intracranial aneurysms.

less-friable aneurysm. The infectious aneurysm wall can be very delicate with a consistency similar to “wet tissue paper,” which increases the risk of intraoperative rupture.

22.1.5 Alternatives to Surgery

The natural history is not well defined for infectious aneurysms due to the relative rarity of the disease and intervention with surgery or endovascular procedures in modern case series. Unruptured infectious aneurysms seem to have a relatively low risk of rupture. Of note, infectious aneurysms were excluded from the International Study of Unruptured Intracranial Aneurysms (ISUIA). Because infectious aneurysms can regress with antibiotic therapy alone, it is reasonable to consider a trial of conservative management for unruptured infectious aneurysms. Conservative management could be considered if a patient is in a moribund state.

Management strategies for infectious aneurysms include both surgery and endovascular procedures, with the latter being first-line treatment in most clinical scenarios. Endovascular procedures can include direct coiling of the aneurysm (with or without balloon/stent assist) or occlusion of the parent artery with coils or liquid embolic agents (NBCA or Onyx). Compared with NBCA, Onyx can be injected more proximally in the parent artery and can be injected multiple times. Stents are generally avoided due to a number of reasons, including the need for dual antiplatelet therapy, the fragile nature of the vessel wall, the hypercoagulable state resulting in increased embolic risk, and the creation of a potential nidus for ongoing infection. If the patient is a poor general anesthetic candidate due to cardiac concerns or other comorbidities, endovascular treatment could be considered under conscious sedation. However, treatment may be more challenging with issues of motion artifact, especially when attempting to navigate a microcatheter into a very distal parent artery. Endovascular treatment also minimizes direct manipulation of the infectious aneurysm, which may decrease the risk of intraoperative rupture. Treatment may potentially be initiated earlier with endovascular procedures compared with surgery, as there is theoretically less need to wait for aneurysm fibrosis. Endovascular treatment

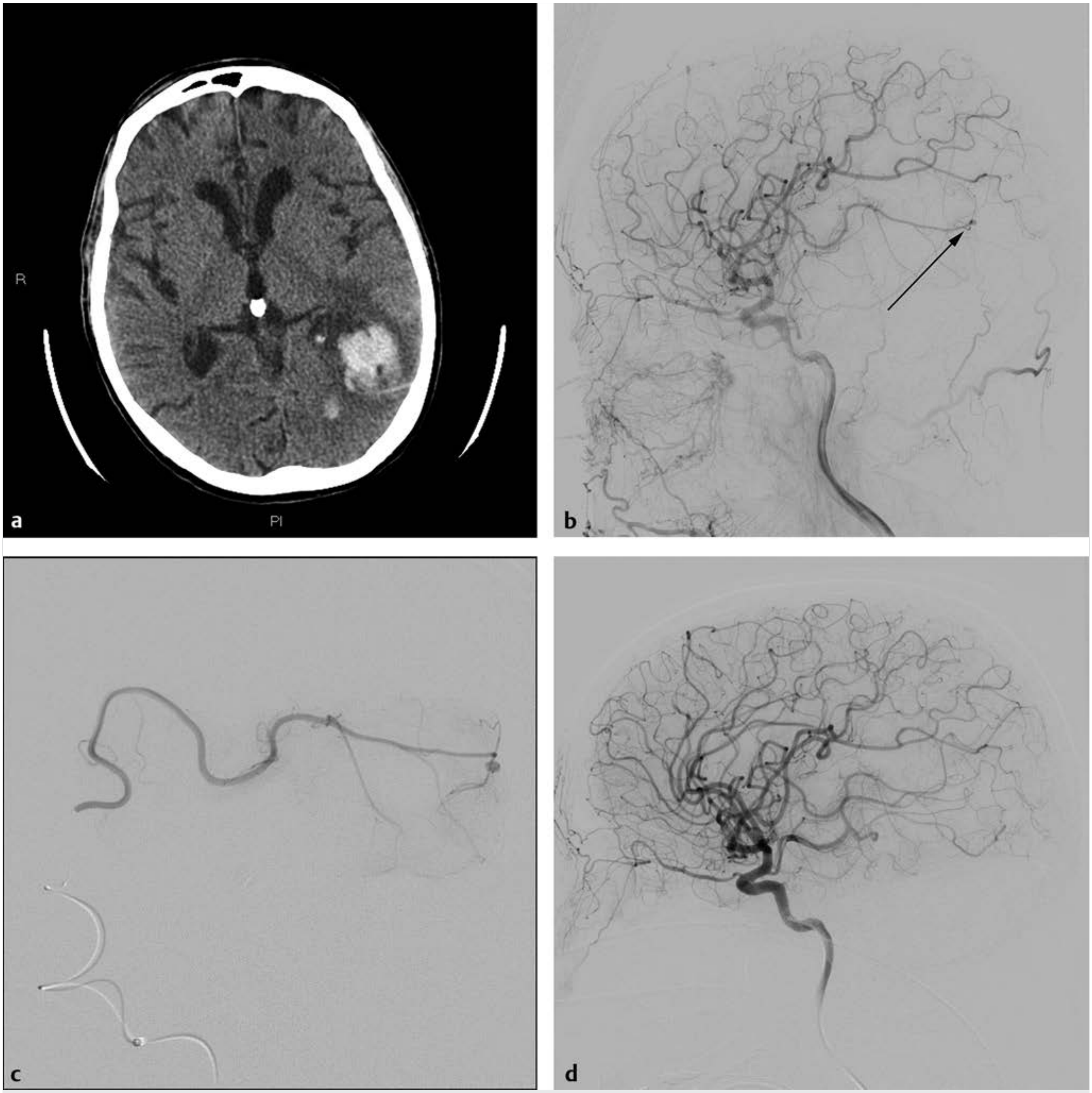

Fig. 22.2 Example of an infectious aneurysm patient treated with endovascular embolization. **(a)** Noncontrast CT head showing a left posterior temporal intracerebral hemorrhage. **(b)** A left lateral internal carotid artery (ICA) injection demonstrating a distal left M3/M4 infectious aneurysm. **(c)** A left lateral superselective injection further delineating the left M3/M4 infectious aneurysm. **(d)** A left lateral ICA injection demonstration obliteration of the aneurysm after embolization with onyx.

also potentially allows earlier heart valve repair/replacement, which requires systemic anticoagulation.

Endovascular Case Example (▶ Fig. 22.1)

A 54-year-old, right-hand dominant, man with known hypertension, and alcohol and cocaine abuse, presented to a peripheral hospital with confusion. Noncontrast CT head demonstrated a left posterior temporal ICH. His hospital course was marked by a non–ST-segment elevation myocardial infarction, alcohol withdrawal seizures, acute kidney injury requiring intermittent hemodialysis, and bacterial endocarditis. Echocardiogram revealed severe mitral valve regurgitation with a 2-cm vegetation. Blood cultures grew streptococcus viridans. He was treated with broad-spectrum antibiotics and later switched to vancomycin after culture sensitivities returned. The patient was then transferred to our institution. CTA was not performed because of the acute kidney injury. MRA revealed no aneurysm. DSA demonstrated a distal left M3/M4 aneurysm, 2 mm in size. This infectious aneurysm was treated with endovascular embolization with Onyx under general anesthetic approximately 3 weeks after the initial ICH. It was felt that the infectious aneurysm was on a quite distal artery suppling a noneloquent territory. The embolization procedure involved a right femoral artery puncture, followed by systemic heparinization and placement of a shuttle catheter in the left common carotid artery. A microcatheter was maneuvered distally from the inferior M2 branch, with the microcatheter tip just proximal to the aneurysm. Onyx was injected with subsequent occlusion of the aneurysm and mild proximal reflux requiring catheter tip detachment. There were no complications from the procedure and patient was transferred back to the peripheral hospital 9 days later with no focal deficits.

22.1.6 Risks of Surgery

Risks of surgery include general risks of undergoing general anesthesia and an operation, including myocardial infarction, pneumonia, deep vein thrombosis, pulmonary embolus, etc. Risks related to the procedure include intraoperative aneurysm rupture, ischemic and hemorrhagic complications, surgical site infection, new neurological deficits, and death. Ischemia can be due to thromboembolic event, prolonged parent vessel occlusion from temporary clips, inadvertent parent vessel occlusion with permanent clips, and bypass thrombosis (if applicable).

22.2 Preoperative Preparation

Management of patients with infectious aneurysms requires a multidisciplinary approach with input from neurosurgery, neurointerventional radiology, infectious disease medicine, cardiac surgery, and critical care medicine. A patient with an infectious aneurysm needs to be stabilized from an airway, breathing, and circulation perspective. Preoperative assessments by general internal medicine and anesthesia are also reasonable to ensure the safety of undergoing a general anesthetic for surgery. Especially for ruptured aneurysms, any coagulopathy should be acutely reversed, including in the setting of a prosthetic heart valve. Routine blood work including complete blood count, electrolytes, creatinine, international normalized ratio/partial thromboplastin time, erythrocyte sedimentation rate, and C-reactive protein should be obtained. Blood and urine cultures are obtained to test for both anaerobic and aerobic bacteria. Consideration can be made for workup for an immunocompromised state by looking at the CD4 count and HIV status. Chest X-ray is needed for preoperative assessment and to rule out a chest infection. A mandible X-ray may identify evidence of dental erosion/abscess. An echocardiogram (transthoracic and/or transesophageal) should be performed to evaluate cardiac and valvular function, to detect valvular vegetations, and to detect a mural thrombus. Noncontrast CT head and CTA (head and neck) are obtained to assess the extent of hemorrhage and the location of the aneurysm(s). Lumbar puncture is not advised in the setting of a ruptured infectious aneurysm. It may be considered if there are ongoing concerns of meningitis with negative blood cultures. If there is radiological evidence of hydrocephalus or the patient presents with decreased level of consciousness after an aneurysm rupture, one can insert an external ventricular drain (EVD) preoperatively. Evaluation of the radial artery should be performed (Allen's test) for potential bypass.

Broad-spectrum intravenous antibiotics should be administered after obtaining blood cultures. Antibiotics can be adjusted based on the type of organism and the culture sensitivities. Generally, intravenous antibiotic treatment lasts for at least 4 to 6 weeks. A peripherally inserted central catheter may be needed early on in the hospital admission. Antipyretics are also administered for fever to maintain normothermia. In ruptured aneurysms, blood pressure is controlled to prevent rebleeding.

Prior to surgical intervention, it is reasonable to obtain a DSA for multiple reasons: (1) assessment of aneurysm morphology and configuration; (2) detection of smaller aneurysms, especially distal ones that may not be detected on CTA; (3) assessment of the caliber of potential bypass donors including the ipsilateral superficial temporal artery (STA) and occipital artery; (4) possibly determining the eloquence of the aneurysm parent artery by performing a Wada test (intra-arterial injection of sodium amytal into the parent artery while the patient is awake); and (5) planning for endovascular treatment with embolization if feasible. For very small distal aneurysms that may be difficult to localize intraoperatively, MR or CTA imaging may be obtained for the purposes of intraoperative neuronavigation.

22.3 Operative Procedure

22.3.1 Positioning and Adjuncts

Preoperative steroids or prophylactic antiepileptic drugs are generally avoided. Mannitol can be used during the craniotomy and prior to the dural opening to help relax the brain. After intubation and initiation of general anesthesia, the supine patient is typically pinned in a fixation headrest apparatus, with the head turned to contralateral side, and roll placed under the ipsilateral shoulder. Adjuncts in the room include the operative microscope, micro-Doppler for intraoperative assessment and regular Doppler to map out the STA on the skin surface, neuronavigation (if available), and indocyanine green (ICG) videoangiography (if available). Intraoperative angiography is also useful if available. The radial artery or greater saphenous vein should be prepped, if needed, for a bypass procedure. We avoid infiltrating the incision line with local anesthetic in ruptured cases.

22.3.2 Incision and Craniotomy

A standard curvilinear incision and pterional craniotomy may be adequate; however, infectious aneurysms are often on distal arteries, so the approach has to be tailored to expose over the aneurysm. The STA is best preserved as well as the frontal and/or parietal branches, depending on the location of the aneurysm. Neuronavigation may be helpful for planning the craniotomy flap in the setting of small distal MCA infectious aneurysms.

22.3.3 Aneurysm Dissection and Clipping

The dura must be opened carefully, especially with distal M3/M4 infectious aneurysms that may be adherent to the dura. After dural opening, the operative microscope is brought into the surgical field. The intraparenchymal hemorrhage is decompressed, if applicable, but not too aggressively as the hematoma may be preventing active aneurysmal bleeding. The aneurysm dissection needs to be performed carefully, by first identifying the afferent artery for proximal control and the efferent branch (es) as well as any perforators near the aneurysm neck and fundus. Temporary clips may be used. Consideration should be given for constructive (clip reconstruction) versus destructive procedures (aneurysm trapping + /– bypass). Often, it is not possible to directly clip the aneurysm due to the friable nature of the wall and fusiform shape of the aneurysm. Therefore, trapping the aneurysm with permanent clips placed proximally and distally can be performed. Intraoperatively, one may see pus or inflammatory material at the site of the infectious aneurysm. Judicious removal of the aneurysm and adjacent infected tissue can be performed especially if the aneurysm is quite superficial (e.g., M3/M4 junction).

More proximal aneurysms may require an intracranial–intracranial bypass or extracranial–intracranial bypass, depending on how amenable the aneurysm is to direct clip reconstruction. Specific bypass techniques are discussed in other chapters. Wrapping the aneurysm, particularly during an intraoperative rupture, is a last resort maneuver in order to reconstruct the parent vessel artery. However, there is a risk that this foreign material may be a nidus for persistent infection or initiate an intense inflammatory reaction such as a "gauzoma."

ICG videoangiography and/or intraoperative angiography may be performed to confirm nonfilling of the infectious aneurysm, although this may be unnecessary when an obvious infectious aneurysm is trapped and resected. Generous irrigation should be performed prior to closure.

22.3.4 Closure

Primary closure of the dura is ideal with avoidance of patch duraplasty if possible. The bone flap usually can be replaced and is not typically infected. Generous bacitracin washes are utilized. The temporalis muscle and fascia are reapproximated with interrupted absorbable 3–0 sutures. The galea is closed with interrupted inverted absorbable 3–0 Vicryl sutures. The senior author prefers skin closure with running 4–0 monofilament absorbable suture.

22.3.5 Operative Case Example (▶ Fig. 22.3)

A 28-year-old, right-hand dominant, male, with known history of intravenous drug use, chronic methadone use, and hepatitis C, presented to our institution with fever and left-sided face and upper limb weakness. Noncontrast CT head demonstrated SAH predominantly in the right sylvian cistern as well as a partial right MCA territory stroke. CTA showed a right superior M2 occlusion but no clear aneurysm. A transthoracic echocardiogram revealed severe aortic valve and mitral valve regurgitation with multiple vegetations measuring as large as 1.5 cm in diameter. Blood cultures grew coagulase-negative staphylococcus. A DSA demonstrated a delayed-filling right MCA bifurcation pseudoaneurysm and confirmed the presence of a thrombosed superior M2. Endovascular treatment may have needed stent assistance, which would require preprocedural dual antiplatelet agents with its associated hemorrhagic risks. Therefore, the patient underwent aneurysm clipping. The patient was intubated and received mannitol for decreased level of consciousness. A right frontal EVD was also placed. Under general anesthetic, a standard right pterional craniotomy was performed with preservation of the STA. The sylvian fissure was split widely, with dissection down to the internal carotid artery (ICA) for proximal control. The right M1 was identified, followed by the two M2 branches. The infectious aneurysm with overlying pus was identified at the right MCA bifurcation site. Intraoperative rupture was controlled with Cottonoids and suction. Temporary clips were placed on the M1 and patent inferior M2. The pseudoaneurysmal sac was dissected off the M1 with overlying fibrotic tissue removed. This revealed a vascular defect at the bifurcation site. A decision was made not to suture the defect due to the friable nature of the wall. Instead, a slightly curved 5-mm aneurysm clip was positioned to cover the occluded superior M2 stump along with the bifurcation, while preserving the M1. ICG videoangiography revealed patent M1 and inferior M2. Intraoperative angiography utilizing the proximal right STA with retrograde injection of contrast down to the right common carotid artery confirmed exclusion of the infectious aneurysm and flow through the right M1 and inferior M2. Aside from the intraoperative rupture, there were no further complications and the patient recovered well.

22.4 Postoperative Management Including Possible Complications

If intraoperative angiography is not available, then a postoperative vascular imaging study should be obtained to confirm aneurysm obliteration. A CT/CTA may be ordered postoperatively to look for any immediate complications, followed by serial DSA or CTA in a delayed fashion until the underlying infection has been adequately treated. Nimodipine may be considered if there is a significant component of SAH but this would be in the absence of any data specifically about its use in infectious aneurysm patients. Intravenous antibiotics are continued for 4 to 6 weeks with input from infectious disease specialists.

Resumption of anticoagulation (e.g., prosthetic heart valve) requires consultation between cardiac surgery and neurosurgery, weighing the risks of postoperative hemorrhage with thromboembolic complications. There are no clear guidelines, so decisions are made on a case-by-case basis. There are also no guidelines regarding the optimal timing of heart valve repair or replacement, which require systemic anticoagulation and hypotension. However, some would recommend cardiac surgery approximately 4 weeks after craniotomy for aneurysm clipping. However, there is a published case report for safe valve repair occurring as soon as 3 days after infectious intracranial aneurysm clipping.

Prognosis for a patient with an infectious aneurysm is poor with a high rate of morbidity and mortality. Once discharged from the hospital, the patient can follow up with neurosurgery in 6 to 8 weeks and follow up with infectious disease medicine as directed. Further follow-up with a noninvasive vascular imaging study can occur at 3 to 6 months with a confirmatory DSA at 1 year to demonstrate continued aneurysm obliteration as well as the absence of de novo aneurysms.

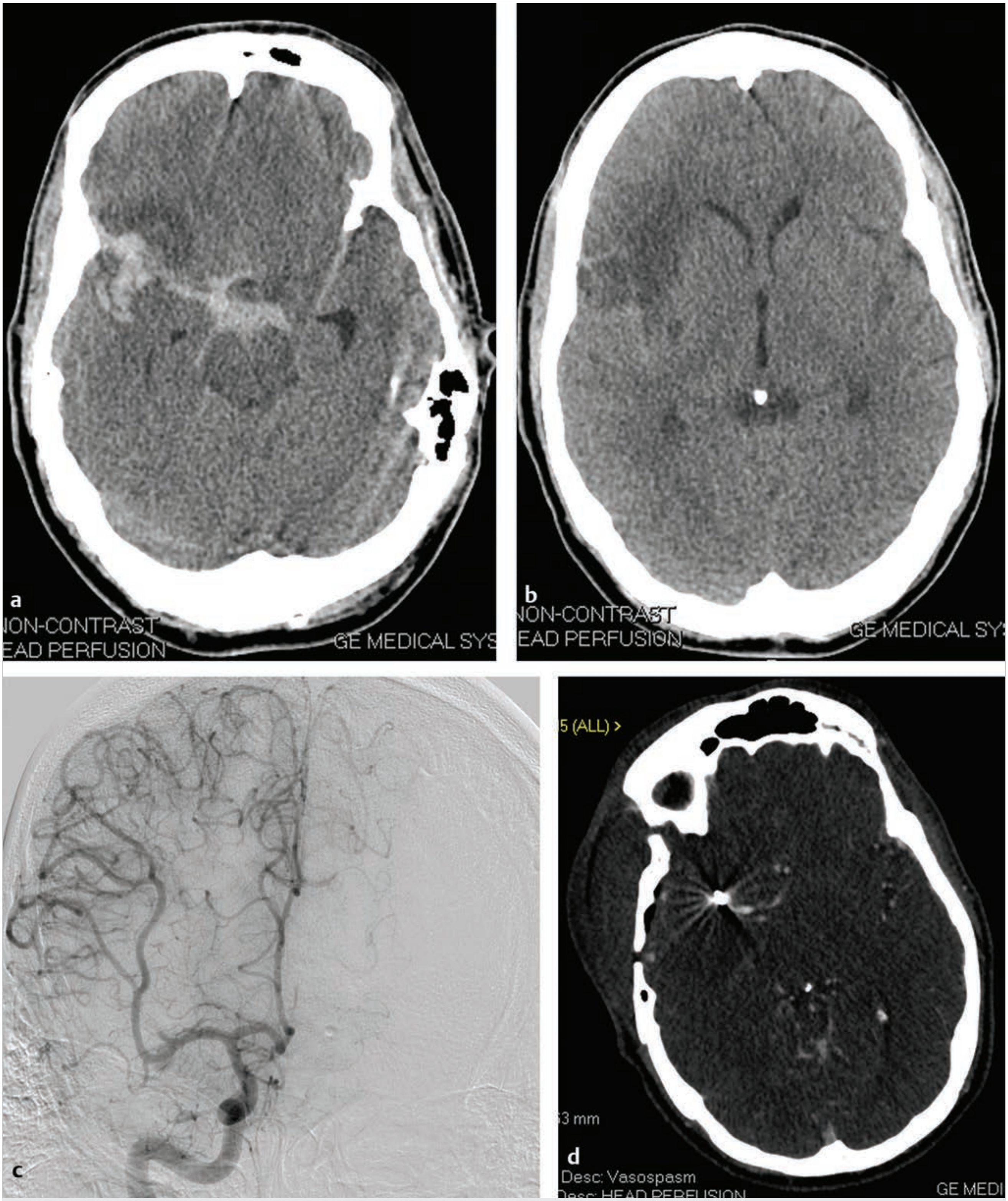
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Fig. 22.3 Example of an infectious aneurysm patient treated with aneurysm clipping. **(a)** Noncontrast CT head showing SAH involving predominantly the right sylvian cistern. **(b)** Noncontrast CT head at a more superior cut showing a partial right MCA territory infarct. **(c)** A right anteroposterior internal carotid artery injection demonstrating a right MCA bifurcation infectious aneurysm pointing inferolaterally as well as a superior M2 occlusion. **(d)** Postoperative CTA demonstrating clip placement and absence of filling of the right MCA bifurcation infectious aneurysm.

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Part II

Vascular Malformations

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23 Arteriovenous Malformations of the Cerebral Convexities

Jacob R. Lepard, John Amburgy, and Winfield S. Fisher III

Abstract

Although most arteriovenous malformations (AVMs) of the cerebral convexity have an inverted conical shape, with the apex pointing toward the ventricle, no two are alike. Two clinical types of AVMs are encountered in practice: unruptured and ruptured. Of the unruptured group, cases are either symptomatic or asymptomatic. ARUBA (A Randomized Trial of Unruptured Brain Arteriovenous Malformations) stresses the important role of medical management for unruptured AVMs. ARUBA has been challenged due to the short time horizon of the study versus the chronicity of AVM natural history (2–4% annual rate of hemorrhage, many times separated by years of no hemorrhage). Sound clinical judgment and strict patient selection coupled with the classical triad of surgical treatment (open surgery, embolization, and stereotactic radiosurgery) are the hallmarks of patient care for all AVMs, be they unruptured or ruptured. Surgical excision begins by controlling the surface feeding arteries in a concentric fashion with care taken to preserve the venous outflow channels. Deeper dissection of the AVM is then undertaken generally in a spiral manner around the AVM, followed by the circumferential dissection of the deep arterial pedicles with eventual control of the small tufts of AVM and then transection of venous drainage. This will allow the AVM to be resected en bloc and minimize hemorrhage and neurologic injury.

Keywords: cerebral convexity, arteriovenous malformation, supratentorial

23.1 Preoperative Planning

23.1.1 Patient Selection

All arteriovenous malformation (AVM) therapies should be directed toward prevention of hemorrhage, neurologic decline, epilepsy, and death. As a general rule, the AVM must be completely obliterated with no evidence of arteriovenous shunting on an angiographic study sometime after the completion of treatment before the AVM can be said to be cured. Thus, pretreatment imaging almost always will include catheter angiography with injection of most, if not all, of the main arteries supplying the head, as well as cross-sectional imaging such as computed tomography (CT) or magnetic resonance imaging (MRI). Additional studies to delineate eloquent brain regions and white matter tracts, such as functional MRI and diffusion tensor imaging, may be indicated.

Approximately 65% of intracranial AVMs are supratentorial; these most commonly present with intraparenchymal hemorrhage and seizures.[1,2] The ideal treatment strategy may involve a combination of observation with medical therapy, surgical resection, embolization, and stereotactic radiosurgery (SRNS), and each or all of these therapies are tailored specifically to each individual lesion.

SRNS has allowed for treatment of lesions previously felt to be too eloquently located for surgical excision. SRNS is most effective in treating AVMs less than 3 cm in diameter, with at best an 80% obliteration in 2 years after treatment.[3] Catheter-directed embolization is rarely curative and is typically used as an adjunct to surgery or SRNS to occlude vascular pedicles or reduce blood flow.[4]

ARUBA (A Randomized Trial of Unruptured Brain Arteriovenous Malformations) supports medical management for unruptured AVMs although the results of this study have been highly controversial for many reasons, including that the highly selected population of unruptured aneurysms probably has a more benign natural history than AVMs that have ruptured, the preponderance of embolization treatment in the treated group, and the short duration of follow-up that will tend to emphasize short-term complications of treatment more than the natural history.[5] Several studies discuss the importance of meticulous patient selection in order to improve the natural history of AVM hemorrhage.[6,7] According to one recently published meta-analysis, unruptured and ruptured AVMs carry a 2.2 and 4.5% annual risk of hemorrhage, respectively.[8] Therefore, it should be emphasized that the primary indication for surgery is prevention of further hemorrhage. Other indications for surgery include: (1) evacuation of the hematoma and (2) control of seizures.

Other considerations for AVM surgery are to have adequate blood available before the surgery, good venous access in case of major bleeding, and good microsurgical instrumentation especially bipolar coagulation forceps such as irrigating, cooled, or specially coated ones to minimize sticking during coagulation.

23.2 Anatomy

Most AVMs have an inverted conical shape, with the apex pointing toward the ventricle, a critical morphological feature of AVMs, which can make surgical excision difficult. Most often, the AVM is separated from the surrounding parenchyma by a gliotic border, hematoma, or ventricular fluid. At the apex, small vascular "tufts" make dissection of the border extremely tedious since these tufts are very fragile, resist coagulation, and bleed extensively when entered. Blood loss can be minimized by dissection around these vascular tufts with control of the small vessels at the apex of the AVM. Draining veins may be single or multiple and may travel to the cortical surface or deep into the subependymal venous system. Preservation of a major draining vein or veins can be critical to smooth excision. If these veins are taken too early, the arterial feeders end in a blind pouch leading to severe AVM expansion, turgidity, and excessive bleeding.[9]

Concomitant feeding vessel aneurysms need to be addressed with open surgery if at all possible. All feeding vessels usually follow normal anatomical patterns, and therefore familiarity of such patterns is paramount with excision strategies. In addition, vessels "en passage" may exist and need to be preserved in order to prevent neurologic deficits.

23.2.1 Intracerebral Hemorrhage and Timing of Surgery

An intracerebral hematoma can create surgical advantages that facilitate AVM resection. The hematoma can create a cavity within the brain that minimizes brain retraction, decreases mass effect, and allows for enhanced visualization of the AVM. Early surgery in these cases can be problematic because the surrounding brain may be edematous and the AVM nidus may be obscured by the hematoma. Although craniotomy for evacuation of the hematoma in the acute phase after hemorrhage may be necessary in patients with a large hemorrhage and significant mass effect, in most cases it is advantageous to delay surgery until the clot liquefies. This process of liquefaction can be followed very easily with CT and/or MRI.

23.3 Operative Procedure

23.3.1 Positioning and Craniotomy

The patient's head should be elevated above the right atrium of the heart to enhance venous return. The head is placed in rigid fixation avoiding extreme rotation and neck extension to prevent jugular vein compromise. The head should be positioned so that the cortical surface of the AVM is parallel to the floor to permit a perpendicular approach to the lesion with minimal brain retraction.

Stereotactic image guidance can help with scalp and bone flap planning. A general principle of AVM surgery is to create a somewhat larger-than-needed bone flap so that there is assurance that one has access to all feeding vessels. When the skin incision is made, patients receive mannitol (0.25 g/kg) and furosemide (10 mg) intravenously to enhance brain relaxation.

23.3.2 Microsurgical Procedure

A circumferential retractor system with flexible, tapered blades is used. A video display linked to the microscope is important to allow the neurosurgical nurses and adjunct staff to view the operation and permit rapid responses during critical times. Intraoperative ultrasound or stereotactic image guidance may be useful. At least three suction devices should be available, and of these, one is assigned for emergency backup.

A complete assortment of vascular microneurosurgical instruments is necessary. Both aneurysm clips and AVM clips should be available. Temporary vessel occlusion can be done with small aneurysm clips, and smaller vessels can be occluded with "Sundt-type" AVM clips. Larger vessels can be permanently occluded with aneurysm clips. To avoid injury to "en passage" arteries, no final commitment or permanent coagulation or ligation is made until the vessel is seen to enter the AVM. Clips allow for reversibility of actions until final commitment is made.

Other required instruments include short and long microscissors and a high-quality nonstick bipolar electrocautery device. The authors prefer platinum tips over irrigating forceps, although both are acceptable. Variable-length instruments allow for initial short depth dissection and, later, deeper dissection.

Opening the dura is not necessarily routine since the feeding arteries, draining veins, or the AVM itself may be adherent to the dura and be at risk for injury, potentially associated with troublesome bleeding in the absence of definition of the AVM anatomy. The dura is opened under loupe or microscope magnification. A circumferential dural incision is made some distance away from the cortical aspect of the AVM to avoid injury to AVM cortical feeding vessels. Under the operating microscope, the cortical surface is inspected for the location of the AVM, feeding arteries, and other superficial vessels. These vessels are dissected and isolated with establishment of the gliotic border between the AVM and cortex (▶ Fig. 23.1). Feeding vessels are followed distally toward the AVM, allowing for placement of temporary vascular clips on feeding arteries, and for permanent occlusion only when the vessel is confirmed to enter the AVM. It may be necessary to open sulci and fissures to identify feeding arteries and to establish proximal control of normal vascular structures. Occasionally, the only part of the AVM visible on the surface of the cortex may be an arterialized cortical vein; this may be followed subcortically to the nidus of the AVM below.

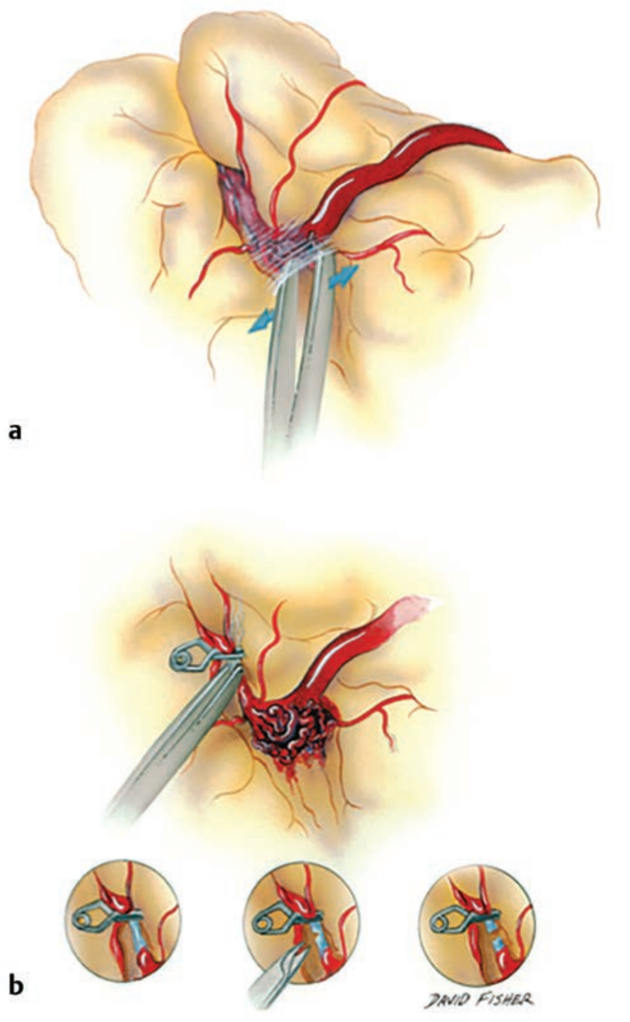

Fig. 23.1 Initial dissection involves proximal control of feeding arteries to the arteriovenous malformation (AVM). **(a)** The arachnoid is opened for exposure. **(b)** Each artery is followed to the AVM, and only when it is seen to enter the AVM is it coagulated and cut. Insets show that each vessel is initially coagulated with bipolar forceps over a length of the vessel and then cut partway to enhance control of bleeding if coagulation attempts have failed. Only after it is found to be fully coagulated is it completely divided.

Dissection of the AVM is continued in the gliotic plane as close as possible to the AVM and should follow the nidus in a circumferential pattern never getting beyond a reasonable dissection plane at all other levels. Arterial pedicles can be difficult to distinguish from arterialized draining veins. A temporary clip can be placed on the vessel to clarify this; a vein will collapse distal to the nidus, whereas an artery will continue to pulsate. Vessels are coagulated over a length of the vessel (at least 1–2 mm) rather than in one focal area. Partial cutting of the vessel with microscissors allows confirmation of occlusion before the vessel is completely transected and the ends of the vessel retract; all vessels are cut in two stages so that if bleeding does occur, control can be accomplished easily (▶ Fig. 23.1). An AVM clip should be applied to vessels that are resistant to coagulation and are robust enough to accept a clip. Feeding arteries “en passage” tend to occur in the fissures and therefore require special attention.

During the dissection, the bipolar forceps are used in coagulation mode with the tips opening parallel to the surface of the dissection cone. Spreading maneuvers should be used cautiously or avoided altogether in order to avoid fragile vessel rupture deep in the resection cavity. Retraction on the AVM can enhance visualization and actually stop oozing just with manual compression. Cotton pledgets can be placed in the plane of the dissection cone to help define it; however, forcing dissection with pledgets is both futile and dangerous. Active bleeding should be stopped when it occurs without packing because multiple bleeding sites can complicate AVM resection exponentially.

Several factors can make an AVM difficult to distinguish from the surrounding brain tissue. Multiple feeding vessels swirling above the AVM may obscure the normal tissue directly underneath. Unruptured AVMs, and AVMs in pediatric patients, are less likely to be surrounded by gliotic tissue. The gliotic tissue along the border of the AVM is usually easily discerned early in the dissection but becomes more difficult as the dissection extends deeper because the size of the feeding vessels becomes literally microscopic. The tenet “if it looks like an AVM, it probably is” can apply in these situations. It can be prudent to err slightly toward the side of the AVM to avoid injury to normal tissue, although too much of a deviation can lead to unforgiving hemorrhage.

As the dissection is carried deeper, the feeding and draining vessels of the AVM become smaller, and small tufts of these vessels may extend from the AVM into the surrounding brain tissue (▶ Fig. 23.2). Substantial bleeding can occur during this phase of the dissection and can be exhausting and difficult to control. Therefore, the perimeter of the dissection cone should be expanded to encompass these small tufts if uncontrolled bleeding is encountered. Focused dissection of these tufts will usually lead to the identification of single vessels during dissection cone expansion (as opposed to the tufts), which allows for coagulation and sharp division of the feeding vessels. Application of AVM clips to these extremely fragile vessels facilitates hemostasis, which may be resistant to coagulation (▶ Fig. 23.2). Errors occur with the premature application of AVM clips on the tufts themselves, which can impede progress just by obscuring the surrounding borders; the AVM clips work best on the solitary feeding vessels by preventing their retraction into normal tissue. AVM clips are also useful during isolation of major feeding vessels that lie deep within sulci; these vessels may also have fragile microscopic perivascular complexes, which defy coagulation.

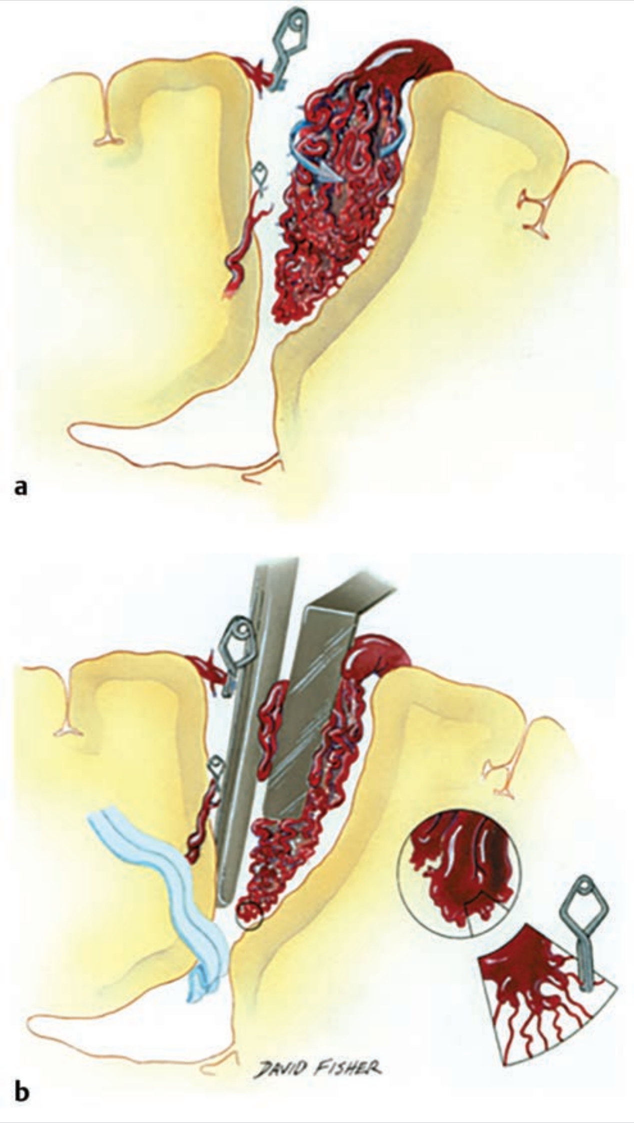

Fig. 23.2 Circumferential dissection of the arteriovenous malformation (AVM). **(a)** Aneurysm and AVM clips enhance control of vessels entering the AVM. **(b)** Tapered retractors are used to minimize brain injury and to follow the dissection cone deeply. Insets show small tufts of AVM extending from the surface of the AVM. Small AVM clips may enhance control of these tufts if the clips are applied appropriately (i.e., on the small feeding vessels and not upon the tuft itself).

After circumferential dissection of the nidus is complete with entry into the ventricle, attention is turned to dissection of the deep arterial feeding vessels. Subependymal feeding vessels lie at the apex of the inverted dissection cone. The ventricle is invariably opened during surgery on larger AVMs; in these cases, a cotton pledget attached to a string is placed within the ventricle to prevent spillage of blood into the ventricular system.

The last step is control and transection of the venous drainage. Major draining veins may still be arterialized after dissection of the nidus due to small arterial feeders that lie underneath or near the venous pedicle. Major draining veins should be left undisturbed until they are first occluded with an aneurysm clip for several minutes and observed (▶ Fig. 23.3). Expansion or increased turgidity of the AVM with temporary

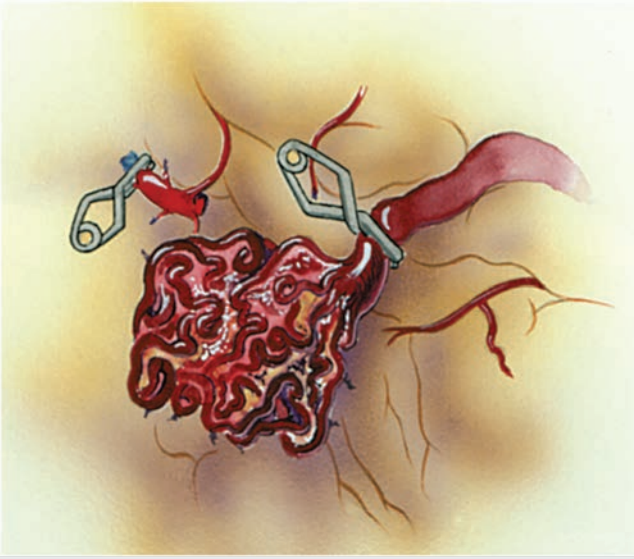

Fig. 23.3 A final clip is placed across the major draining vein, as a final test that the AVM is ready for removal. The clip is left in place for several minutes and may be removed if problems arise.

occlusion of the draining veins is a sign that arterial pedicles still exist and need to be found prior to permanent occlusion of the draining vein. Attention should be directed toward the area near the draining veins; frequently, persistent feeding vessels may be found there or deep within the ventricle. The AVM should then be lifted out of the brain en bloc. Refer to **Video 23.1** for demonstration of operative technique. Partial resection across an AVM will lead to hemorrhage that is difficult to control at best and almost impossible at worst.

After removal of the AVM, the cavity is irrigated and meticulously inspected for evidence of residual nidus. A brief Valsalva maneuver can confirm that adequate hemostasis has been achieved and that the resection is complete. If hemostasis seems tenuous at the completion of the resection, a ventricular catheter can be placed in the ventricle or adjacent to the resection cavity to allow for drainage and pressure monitoring in the immediate postoperative period. “Testing” the cavity with hypertension is never warranted, and operative hypotension may compound neurologic deficits. Some surgeons employ intraoperative angiography to confirm the extent of AVM resection.

23.4 Postoperative Management Including Possible Complications

All patients should be observed in the neurosurgical intensive care unit for at least 24 hours. Blood pressure is maintained at, or slightly below, the patient’s baseline. Adequate hydration should be provided to minimize the risk of intracranial venous thrombosis. The primary postoperative concern following surgical resection is hemorrhage, which can usually be attributed to residual AVM, venous thrombosis, or normal perfusion pressure breakthrough.[10] Residual AVM may require return to the operating room for resection or, under some circumstances, treatment by radiosurgery or endovascular means. Generally, catheter angiography at least several days after the surgery is required to document complete obliteration of the AVM.

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24 Arteriovenous Malformations of the Basal Ganglia and Thalamus

Jeremiah N. Johnson, Michael Lim, Mario Teo, and Gary K. Steinberg

Abstract

Arteriovenous malformations (AVMs) of the basal ganglia and thalamus represent around 10% of all AVMs. Due to their deep eloquent location, AVM hemorrhage can result in profound neurological deficits. Their location, however, makes safe treatment very challenging. Advances in interventional neuroradiology, microneurosurgery, and stereotactic radiosurgery have increased the number of these vascular malformations that can be treated successfully.

Keywords: arteriovenous malformation, basal ganglia, thalamus, intracerebral hemorrhage

24.1 Patient Selection

The overall risk of hemorrhage for cerebral arteriovenous malformations (AVMs) is about 2 to 4% per year.[1] Annual rupture rates are lower for unruptured AVMs compared to patients with prior AVM bleed at 2.2 versus 4.5%, respectively.[2] Hemorrhage rates for AVMs in the thalamus and basal ganglia may be higher. In a review of untreated, patients with > 500 patient-years of follow-up who were ultimately referred to Stanford for evaluation, the pretreatment annual rupture rate was 9.8% per year,[3] and deep AVM location has also been identified as an independent risk factor for hemorrhage in large natural history studies.[1,2] Periventricular location and deep venous drainage have been shown to increase the risk of bleeding of cerebral AVMs, and a history of previous hemorrhages correlates with hemorrhage recurrence for AVMs of the thalamus and basal ganglia. Hemorrhage from a basal ganglia or thalamic AVM also carries a risk of serious morbidity, with up to 85% of such cases developing hemiparesis or hemiplegia. The higher risk of hemorrhage and greater morbidity from hemorrhage should be factored into the decision as to whether to treat basal ganglia and thalamic AVMs. However, safe surgical access to the thalamus and basal ganglia is limited, and morbidity from treatment complications in these eloquent locations can be similarly devastating, making the assessment of risk–benefit challenging.

An important factor in patient selection is whether the AVM and/or any hemorrhage from it reaches a pial or ventricular surface. In such cases, if one can expose the AVM from that surface, then resection is at least theoretically possible. If no pial or ventricular surface is reached, then the approach has to transgress brain tissue, the function of which has to be potentially expendable without causing morbidity. There are several surgical corridors that can be used to access these deep-seated lesions. Small AVMs of the medial thalamus that are accessible through the lateral ventricle can be resected with minimal morbidity. AVMs limited to the pulvinar can often be removed safely. AVMs in the anterior thalamus or basal ganglia presenting to the insular region can be accessed through a transsylvian exposure with care taken to spare the motor fibers traversing the posterior limb of the internal capsule. Oftentimes, a multimodal approach employing embolization and radiation followed by subsequent delayed microsurgery can be used. If the lesion is large and does not present to a ventricular or pial surface, one may consider either or both stereotactic radiosurgery and embolization.

Poor surgical candidates include patients with severe comorbidities, elderly patients, and patients with devastating neurological deficits. Patients with lesions located in the posterior limb of the internal capsule are also excluded because of the high risk of permanent deficits. In patients with asymptomatic basal ganglia and thalamic AVMs, the risk of surgical morbidity and other patient-specific characteristics should be carefully weighed against the natural history of the lesion and patient-specific factors, such as lesion location, patient age, comorbidities, angiographic features, and history of hemorrhage. Patients presenting with hemorrhage are known to have a worse natural history and poorer outcomes after rehemorrhage; thus, carefully tailored low-morbidity treatments with a goal of AVM obliteration can decrease future hemorrhage risk and the associated morbidity.

24.2 Preoperative Preparation

24.2.1 Imaging

Preoperative magnetic resonance imaging and catheter angiograms are essential to understanding AVM features, anatomy, and location. In some cases, preoperative diffusion tensor imaging can be useful to localize nearby traversing motor tracts and understand their position relative to the nidus, aid in surgical approach selection, and enhance the intraoperative neuronavigation. We highly recommend the use of neuronavigation systems to guide the approach and resection of deep AVMs.

24.2.2 Embolization

On traditional catheter angiography, these AVMs are generally fed from the medial and lateral lenticulostriate arteries, recurrent artery of Heubner, thalamogeniculate arteries, thalamoperforating arteries, and anterior and posterior choroidal arteries. Almost all AVMs in the basal ganglia and thalamus have deep venous drainage. In patients with AVMs amenable to embolization, we recommend staged embolizations spaced at least 1 week apart to reduce the volume of AVM and potential bleeding during surgery. We never attempt to embolize more than 30% of the AVM at any session because more aggressive embolization can cause swelling and hemorrhage.

24.2.3 Anesthetic Technique

Surgery is performed under general endotracheal anesthesia. The anesthesiologist should control the patient's mean arterial pressure (MAP) between 70 and 80 mm Hg throughout induction and surgical opening, and at 60 to 70 mm Hg during resection of the AVM through emergence from anesthesia. We also recommend mild hypothermia with a target core temperature of 33 °C to 34 °C achieved via a cooling blanket or femoral venous catheters.

24.2.4 Monitoring

Electrophysiological monitoring and mapping are important tools in resection of AVMs. The use of continuous bilateral upper and lower somatosensory evoked potentials and motor evoked potentials along with cortical mapping can be invaluable in decreasing the risks associated with the surgical and endovascular management of these lesions.

24.2.5 Additional Preparation

Prior to surgery, we sometimes use a ventriculostomy or lumbar drain for brain relaxation prior to positioning. However, this is not necessary if an intraventricular approach is used because cerebrospinal fluid (CSF) can be drained directly with opening of the ventricle. Additional steps to induce brain relaxation include hyperventilation or diuresis or both. The groin should also be prepared for possible intraoperative angiography.

24.3 Operative Procedure

24.3.1 Operative Positioning and Exposure

There are five main surgical approaches used individually or in combination: frontal, interhemispheric transcallosal, parietal interhemispheric transcallosal, occipital transtentorial infrasplenial, supracerebellar infratentorial, and transsylvian and transcortical (frontal or parietal) (▶ Fig. 24.1 and ▶ Fig. 24.2). The approach is determined by the location of the lesion. AVMs of the caudate are approached through a frontal interhemispheric transcallosal exposure (▶ Fig. 24.3), whereas AVMs in the thalamus are exposed through a parietal interhemispheric transcallosal approach (▶ Fig. 24.4 and ▶ Fig. 24.5).

24.3.2 Surgical Technique

Adequate exposure is critical to resecting deep vascular malformations. After the vascular malformation is exposed and identified, microneurosurgical techniques are employed to remove the AVM. In removing a deep AVM, one must identify the major arterial feeders as early as possible. These can be predicted from the preoperative angiogram, and neuronavigation can aid in localizing them intraoperatively. The arterial feeders should then be divided close to the nidus while preserving the draining veins. We recommend exposing several millimeters of a feeding artery prior to coagulation or using microclips because of its tendency to retract into the parenchyma after cutting. If the retracted vessels are incompletely coagulated or spontaneously rebleed, they can require further dissection into eloquent areas to regain hemostasis. The surgeon must be careful not to confuse arterialized veins with feeding arteries. The nidus is then dissected from the surrounding brain as the surgeon looks for gliotic and hemosiderin-stained brain as a plane. We emphasize dissecting close to the nidus to minimize the risk of injuring normal brain and to avoid entering the AVM. The final step is to divide the draining veins. Careful hemostasis should be achieved before closure.

24.3.3 Interhemispheric Transcallosal

The patient is positioned supine with the head slightly elevated above the heart and flexed 20 to 30 degrees (▶ Fig. 24.2). Alternatively, it is sometimes helpful to position the patient in lateral or three-quarter prone position with the right side down, which allows the right hemisphere to fall away from the falx, facilitating the exposure along the interhemispheric fissure. A paramedian trapdoor incision is made on the same side as the AVM. For the frontal interhemispheric transcallosal approach, we make two-thirds of the flap anterior and one-third posterior to the coronal suture. For the parietal approach, the anterior

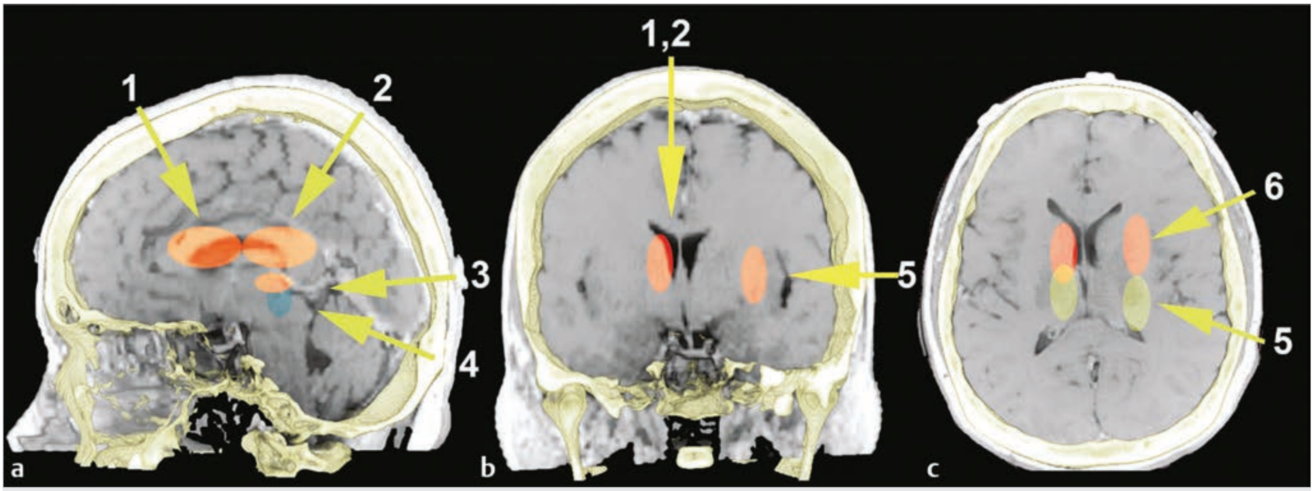

Fig. 24.1 (a–c) Approaches to basal ganglia and thalamic arteriovenous malformations. The translucent oval areas indicate the anatomical location of the lesion and the arrows show the corresponding approach. (1) Frontal interhemispheric transcallosal. (2) Parietal interhemispheric transcallosal. (3) Occipital transtentorial infrasplenial. (4) Supracerebellar infratentorial. (5) Transsylvian. (6) Transcortical (**Video 24.1**).

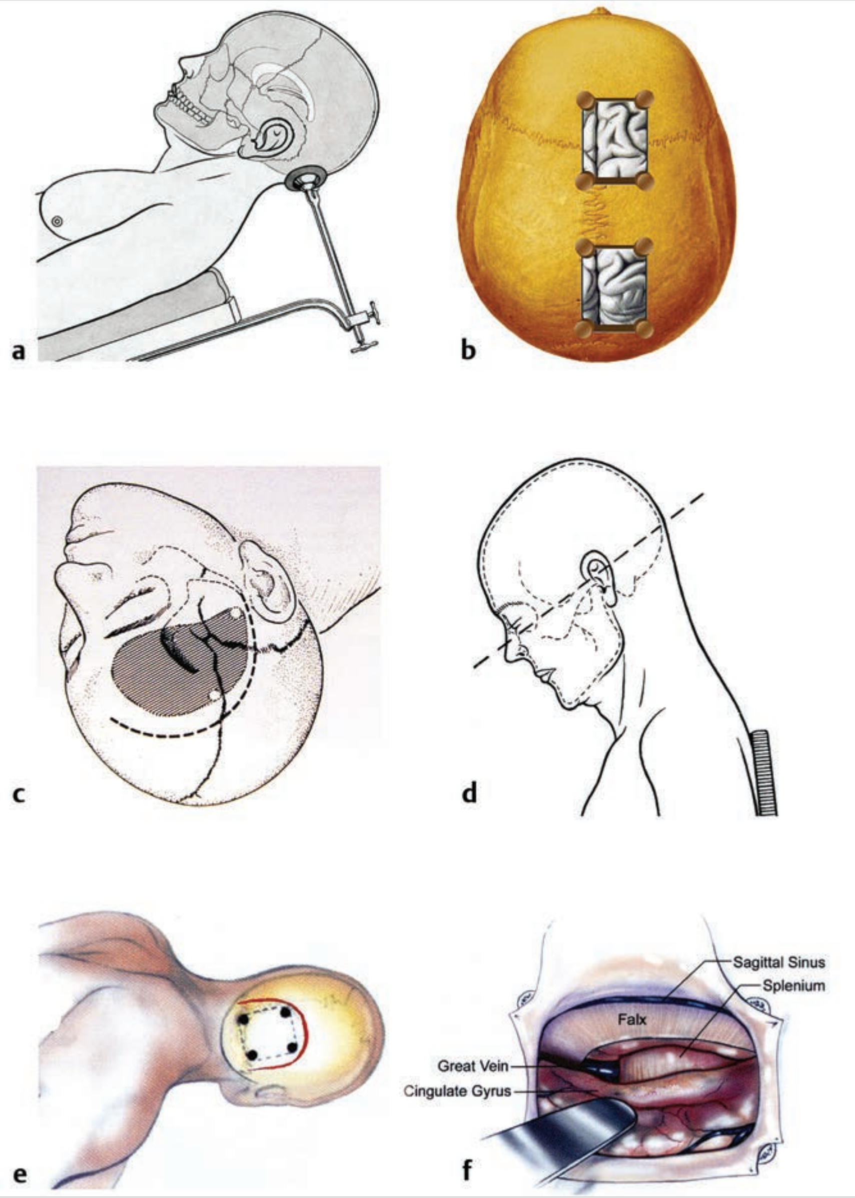

Fig. 24.2 Patient positioning, incision, and bone flap for the different approaches. **(a)** For the frontal and parietal interhemispheric transcallosal approach, the patient is positioned supine, and the head is flexed 20 to 30 degrees and elevated above the heart. Alternatively, the patient can be positioned in a lateral or three-quarter prone position with the right side down, which allows the right hemisphere to fall away from the falx. **(b)** A trapdoor skin incision over the midline is performed, and the craniotomy is placed one-third behind the coronal suture and two-thirds in front for the frontal approach or with the anterior craniotomy border behind the postcentral gyrus for the parietal approach. **(c)** Frontotemporal craniotomy for the transsylvian approach is done with the patient supine and the head rotated 20 degrees and extended. **(d)** The supracerebellar infratentorial approach is done in the sitting position, allowing the cerebellar hemispheres to fall down with gravity. Alternatively, the modified prone Concorde position can be used. Slight flexion of the head opens the access to the posterior fossa, but too much flexion has to be avoided so as not to impair venous outflow. **(e)** Three-quarter prone position is chosen for an occipital transtentorial infrasplenial approach. **(f)** This position allows the occipital lobe to fall away from the falx, with gravity limiting the need for retraction and opening the view to the ambient and quadrigeminal cisterns.

margin of the craniotomy should be posterior to the postcentral gyrus. The bone flap is made large enough in the anteroposterior direction to allow preservation of cortical draining veins when entering the interhemispheric space. It is taken over the midline, exposing the superior sagittal sinus. The dura is incised with a base toward the sinus and tacked up to be pulled gently to the left side, giving a wider access to the interhemispheric fissure. Cortical veins should be preserved. When large cortical draining veins prevent exposure from the right, a dural incision and approach may need to be performed on the left side. If the brain is tight, either or both mannitol and furosemide are given to promote diuresis, and the patient is hyperventilated to increase brain relaxation. Once achieved, retractors are used to visualize the corpus callosum, and the operating microscope is brought into the field. Care must be taken to avoid overly aggressive retraction on the medial frontal lobe because lower limb weakness can occur from this maneuver. The callosomarginal and pericallosal arteries are identified (▶ Fig. 24.3**g**). The pericallosal arteries should be separated in the midline. A callosotomy is then performed using bipolar electrocautery and small-bore suction to enter the lateral ventricle. Image guidance is employed to help determine the site of callosotomy and then the location of the AVM in the caudate or thalamus (▶ Fig. 24.4**g**). If the AVM is not on the surface of the thalamus, an approach into the thalamus through the pulvinar (posterior part of the thalamus) can be performed. The AVM is then resected using the microsurgical principles described in the beginning of this section.

24.3.4 Transsylvian Approach

For AVMs located in the putamen and extending to the insular cortex, a transsylvian approach is used. A classical frontotemporal craniotomy is performed, exposing the sylvian fissure (▶ Fig. 24.2). Using neuronavigation, the shortest distance of the AVM to the superficial sylvian fissure is defined and the fissure is opened (▶ Fig. 24.6). The difficulty is to differentiate AVM feeding from nonfeeding middle cerebral artery branches. Typically, the middle cerebral branches have to be skeletonized

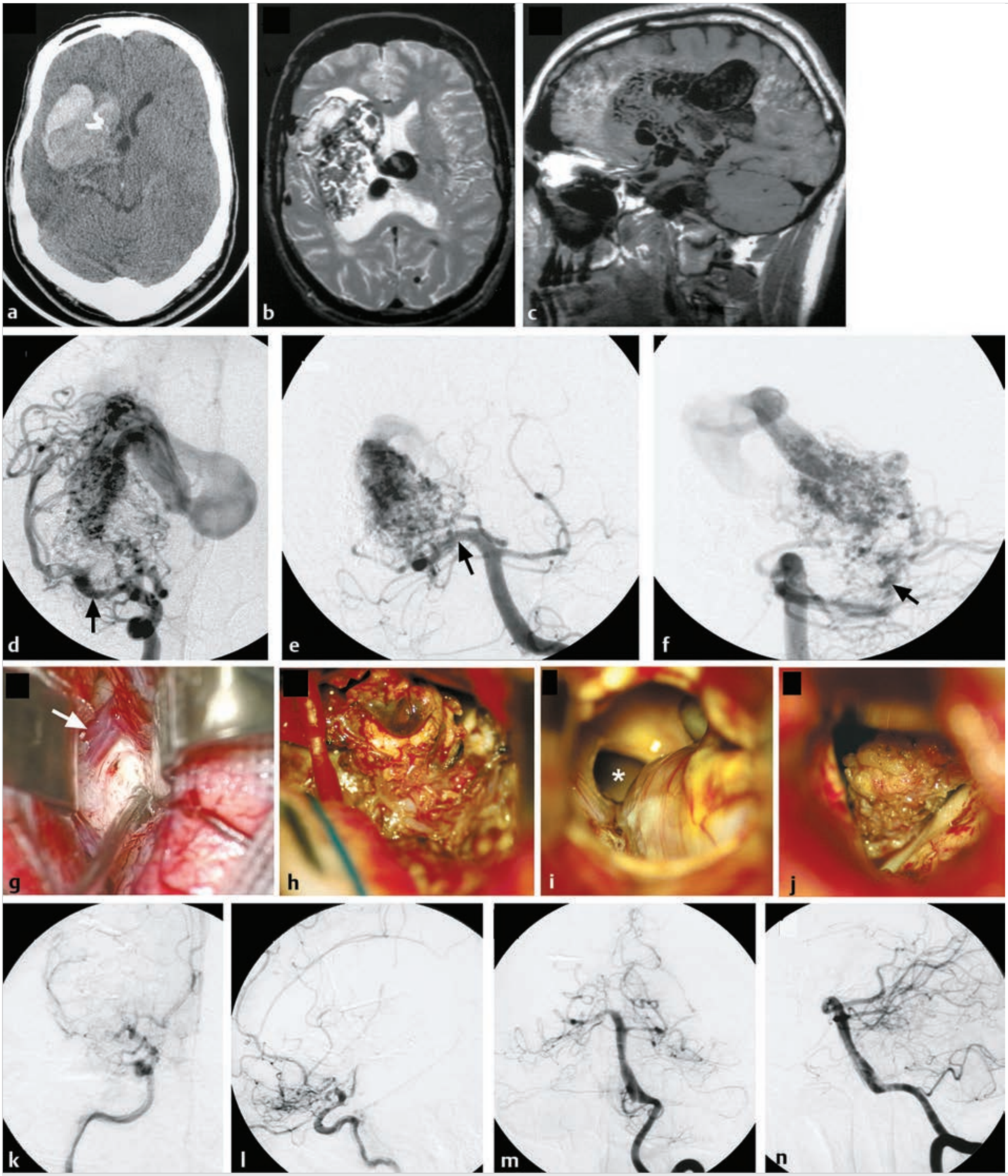

Fig. 24.3 **(a)** A 28-year-old male presented with intracerebral hemorrhage from a 6-cm large, right basal ganglia/thalamic arteriovenous malformation (AVM) after unsuccessful proton beam radiation therapy 8 years prior. **(b)** Typical flow voids are seen in the T2 axial and **(c)** sagittal magnetic resonance imaging (MRI). After four embolization sessions, the AVM flow was reduced by 75%. The remaining AVM was fed by branches of the right middle cerebral artery (MCA) **(d)** (*arrow*, right ICA injection), right lateral lenticulostriates (*arrow*), and anterior **(e)** and posterior **(f)** thalamoperforators (*arrow*, vertebral artery injection) and had both superficial and deep drainage. A 1-cm intranidal aneurysm was also found. Following the embolizations, staged surgery was performed using three separate approaches. **(g)** First, a frontal interhemispheric transcallosal approach was used to resect the anterior and medial portion of the AVM. Special care was taken to leave draining veins intact. The interhemispheric fissure with the pericallosal arteries is seen (*arrow*). **(h)** The AVM surface was exposed, and this portion of the AVM was subsequently resected. **(i)** View into the lateral and third ventricle (*asterisk*) after resection of the AVM. **(j)** In a second stage, a parietal transcortical approach was used to resect the posterior and lateral aspect of the AVM. A third operative stage used a frontal transcortical exposure. **(k)** Postoperative anteroposterior and **(l)** lateral right internal carotid and anteroposterior **(m)** and lateral **(n)** vertebral injections show complete resection of the AVM (**Video 24.1**).

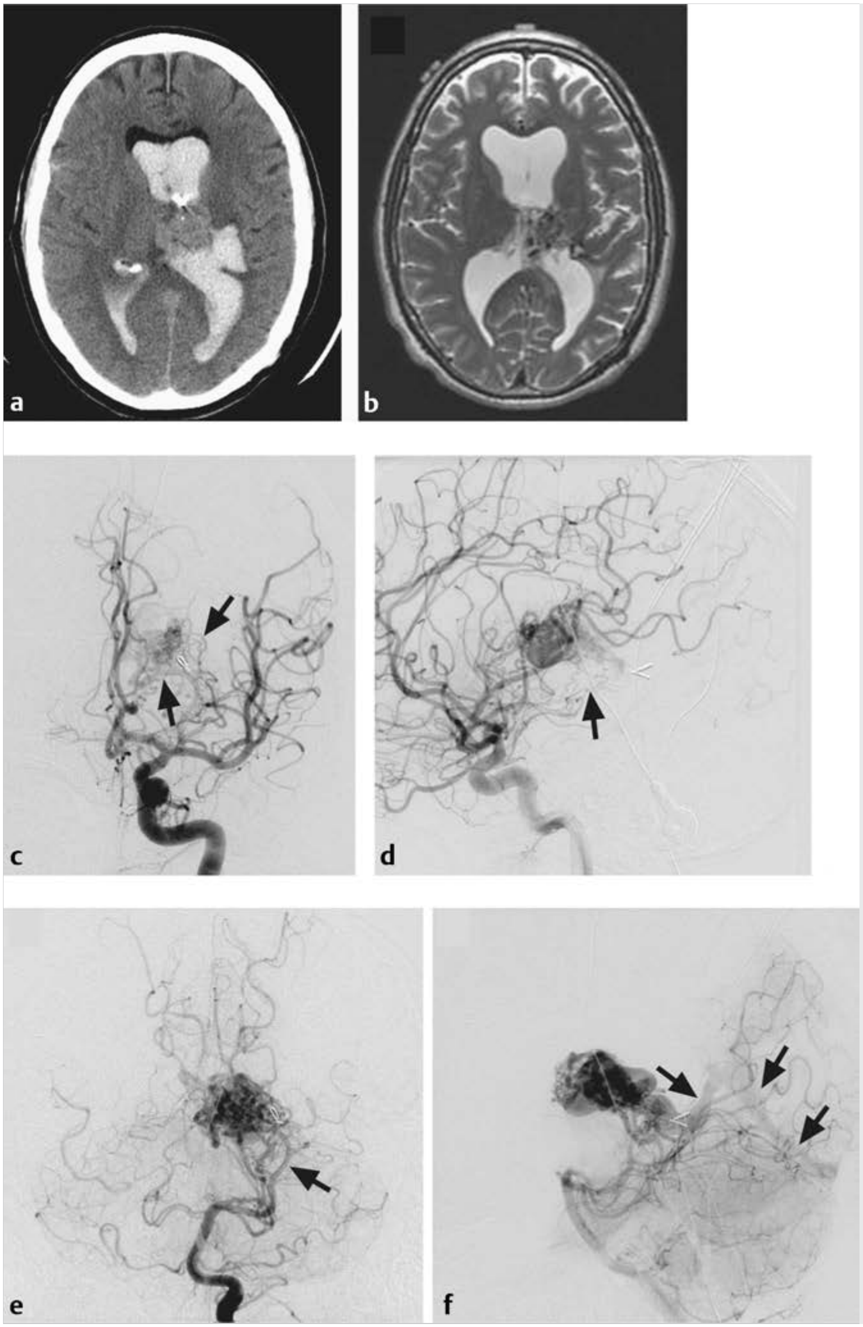

Fig. 24.4 **(a)** A 45-year-old male presented with intraventricular hemorrhage on computed tomography (CT) scan from thalamic arteriovenous malformation (AVM) partially treated 24 years earlier. **(b)** T2-weighted magnetic resonance image showing the thalamic AVM with an intraventricular component. **(c)** The AVM was fed by the left middle and anterior cerebral artery branches (*arrows*), **(d)** the anterior choroidal artery (*arrow*), and **(e)** the left posterior cerebral artery (*arrows*). **(f)** The AVM drained deep into the vein of Galen and straight sinus (*arrows*).

for one to appreciate the anatomy and avoid coagulating a normal traversing or "en passage" branch. The AVM is resected, taking the draining veins at the end (▶ Fig. 24.6 and ▶ Fig. 24.7).

24.3.5 Other Approaches

For AVMs located in the lateral basal ganglia (▶ Fig. 24.3) or thalamus (▶ Fig. 24.5 and ▶ Fig. 24.8), not extending to the insular cortex, a transcortical approach can be considered (▶ Fig. 24.3**j**). Image guidance is used to define the nearest cortical entry zone to reach the AVM. Ultrasound can also be used to localize an AVM when one is approaching it transcortically. Microsurgical techniques are used as described above.

For lesions in the pulvinar, an occipital transtentorial infrasplenial approach is chosen. The patient is positioned in a lateral or three-quarter prone position (▶ Fig. 24.2**e**) to allow the occipital lobe to fall away from the falx by gravity, minimizing the need for retraction. The skin and bone flap are performed, exposing the superior sagittal sinus. The dura is opened with its base toward the sagittal sinus. Usually, there are no bridging veins and the occipital lobe can be retracted readily. The tentorium can be divided lateral to the straight sinus, from the free edge of the incisura back to the transverse sinus if needed, giving access to the ambient and quadrigeminal cisterns. Cutting the tentorium to widen the exposure also facilitates working around the vein of Galen and the internal

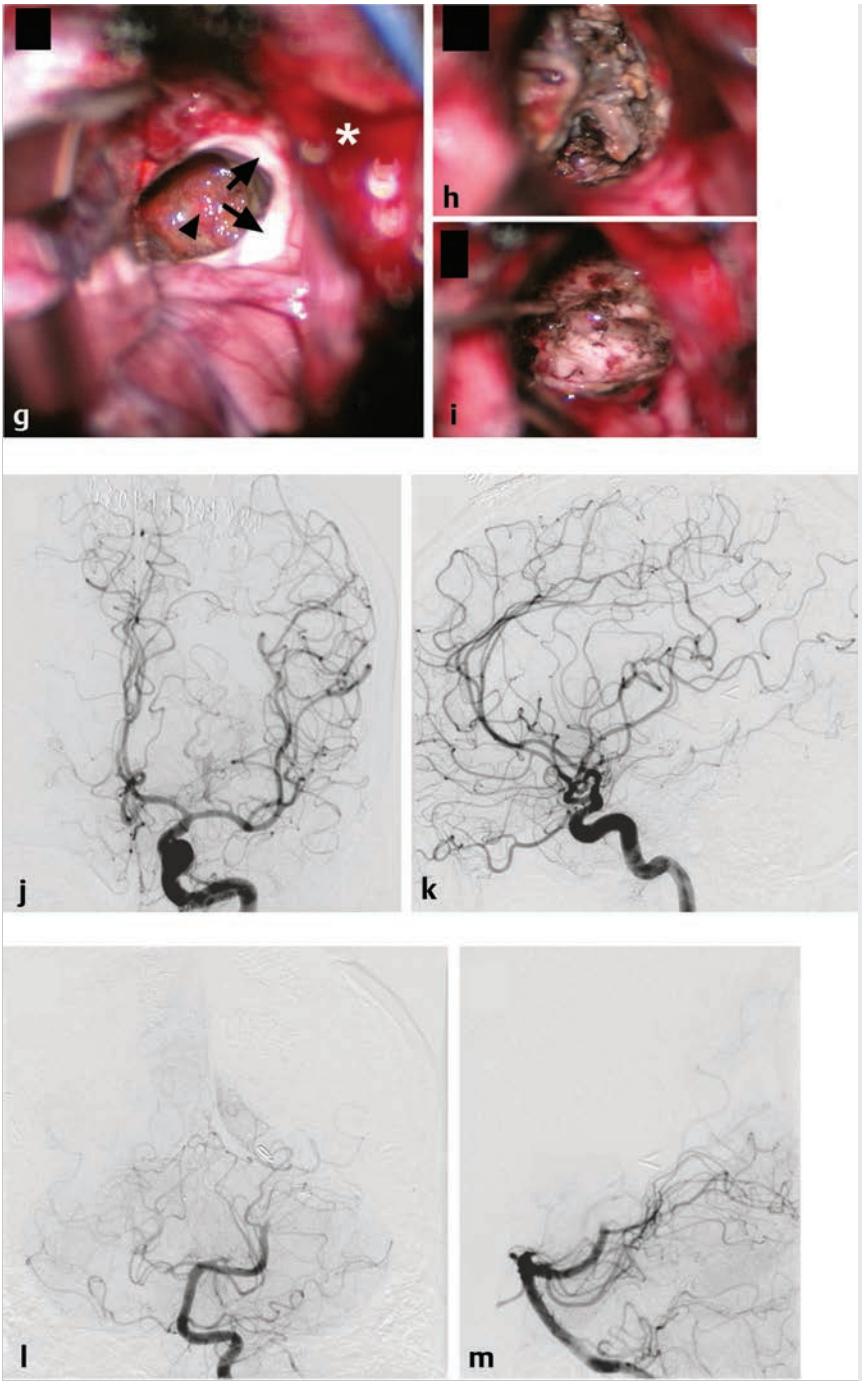

Fig. 24.4 (*Continued*) **(g)** A left parietal interhemispheric transcallosal approach was performed. The asterisk shows the midline, and the corpus callosum (*arrows*) has been incised to access the lateral ventricle and the thalamus. Image guidance with microscope integrated laser pointer (*arrowhead*) was used to determine the entry point into the thalamus. **(h)** The AVM was exposed and **(i)** resected. **(j)** Postoperative anteroposterior and **(k)** lateral left internal carotid artery and anteroposterior **(l)** and lateral **(m)** vertebral angiograms show complete resection of the AVM.

cerebral veins (▶ Fig. 24.2**f**). The AVM is approached through the pulvinar using image guidance to localize the lesion, and resection is performed as previously described.

AVMs extending from the pulvinar into the midbrain can be accessed via an infratentorial supracerebellar approach (▶ Fig. 24.9). The patient is positioned in the semi-sitting position with the head above the heart and chin tucked down slightly (▶ Fig. 24.2**d**). Too much flexion can impair venous outflow. Alternatively, the modified prone Concorde position can be used. A midline skin incision from the inion to C2 is performed, and the neck muscles are dissected in the midline. The suboccipital bone is exposed down to the foramen magnum, and the arch of C1 is identified. The torcula is located using image guidance. A standard suboccipital craniotomy is performed. The craniotomy extends a little over the transverse sinus to allow retraction of the tentorium and therefore opening of the supracerebellar space. The dura is opened in a **V** shape and tacked up, pulling the transverse sinus cranially. The cerebellar hemispheres fall down after opening the arachnoid and draining the CSF. Bridging veins are usually encountered along the midline, draining into the straight sinus, and laterally above the cerebellar hemispheres, draining into venous lakes of the tentorium. The midline veins are slightly stretched, coagulated near the cerebellar surface, and cut, allowing access to the quadrigeminal cistern and pineal region. The internal cerebral veins, the

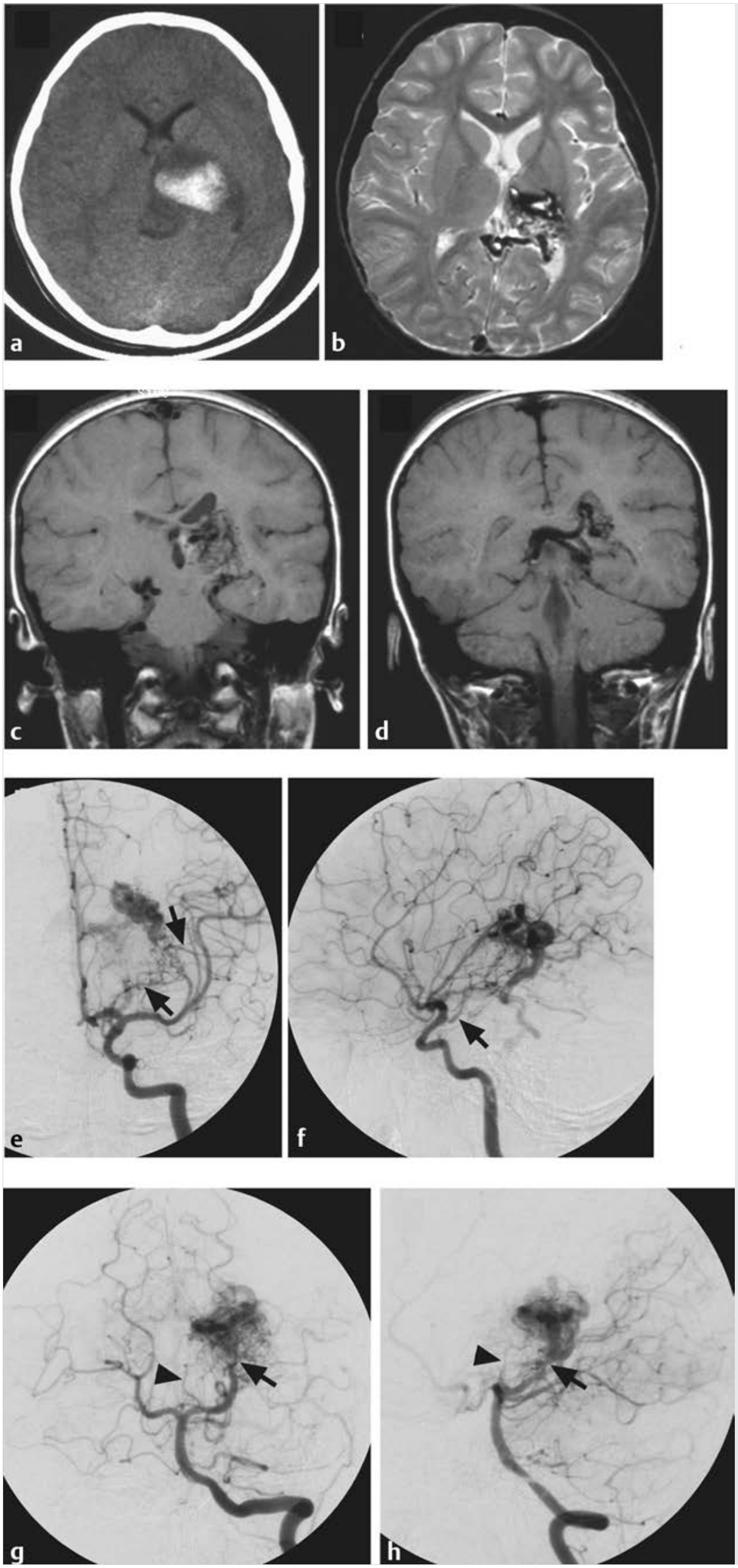

Fig. 24.5 A 7-year-old girl presented with hemiparesis and dysphasia after intracerebral hemorrhage. **(a)** Computed tomography (CT) scan from a left thalamic and basal ganglia arteriovenous malformation (AVM). **(b)** T2 axial and **(c, d)** T1 coronal magnetic resonance scans. **(e)** The AVM was fed by left lenticulostriate, left middle cerebral (*arrows*), **(f)** anterior (*arrow*), and posterior choroidal (*arrow*) arteries and posterior thalamoperforators (*arrowhead*) seen on anteroposterior **(g)** and lateral **(h)** vertebral artery angiograms.

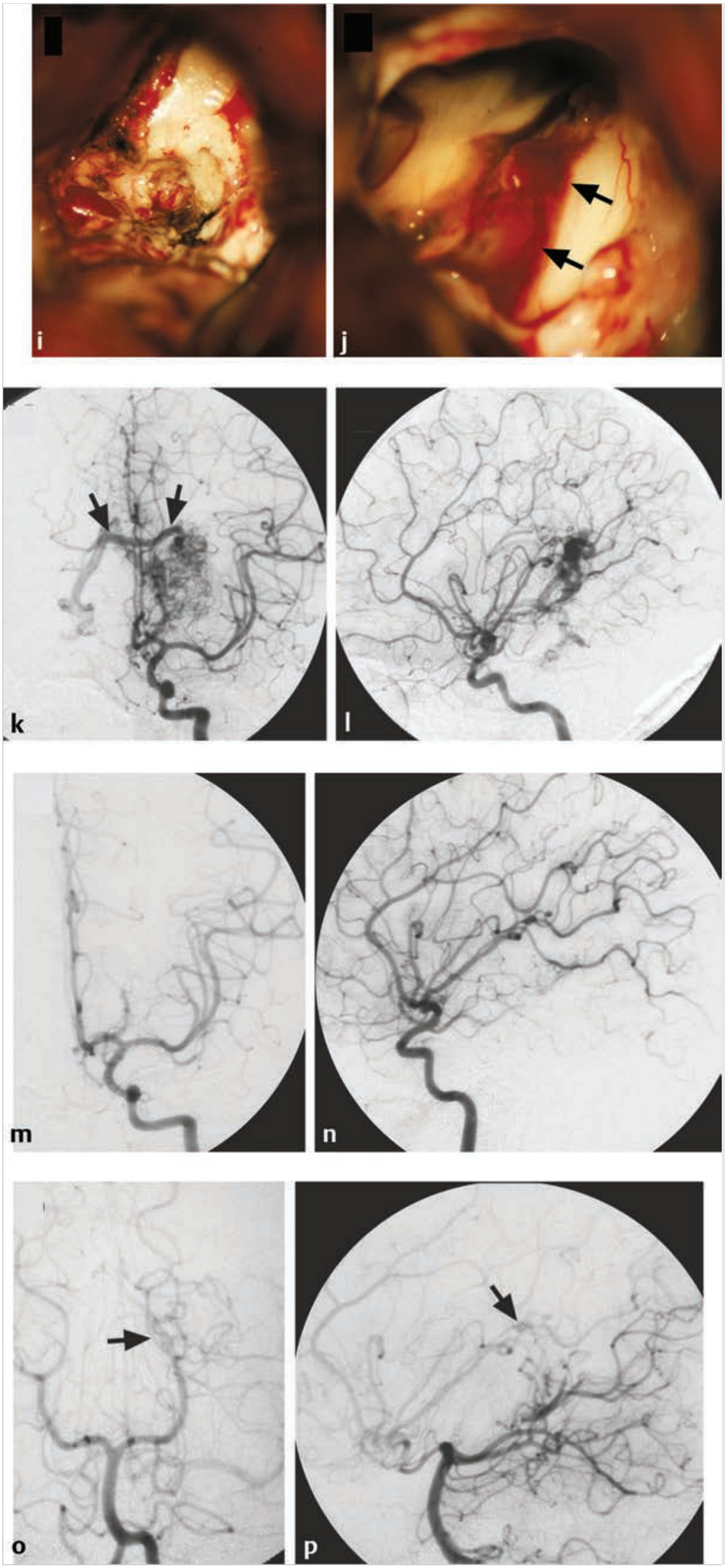

Fig. 24.5 (*Continued*) **(i)** A staged surgery was performed resecting the medial thalamic portion through a left parietal interhemispheric transcallosal approach (view of the thalamus and the AVM). **(j)** Care was taken to leave the anteriorly draining vein on the thalamic surface intact (*arrows*). The anteroposterior **(k)** and lateral **(l)** internal carotid artery angiogram after the first surgery demonstrates the intact draining vein (*arrows*). The more lateral basal ganglia portion was resected via a parietal transcortical approach. **(m)** Postoperative anteroposterior and **(n)** lateral right internal carotid angiograms show no residual but the anteroposterior **(o)** and lateral **(p)** vertebral angiograms demonstrate a tiny residual AVM (*arrow*), which was subsequently treated with stereotactic radiosurgery.

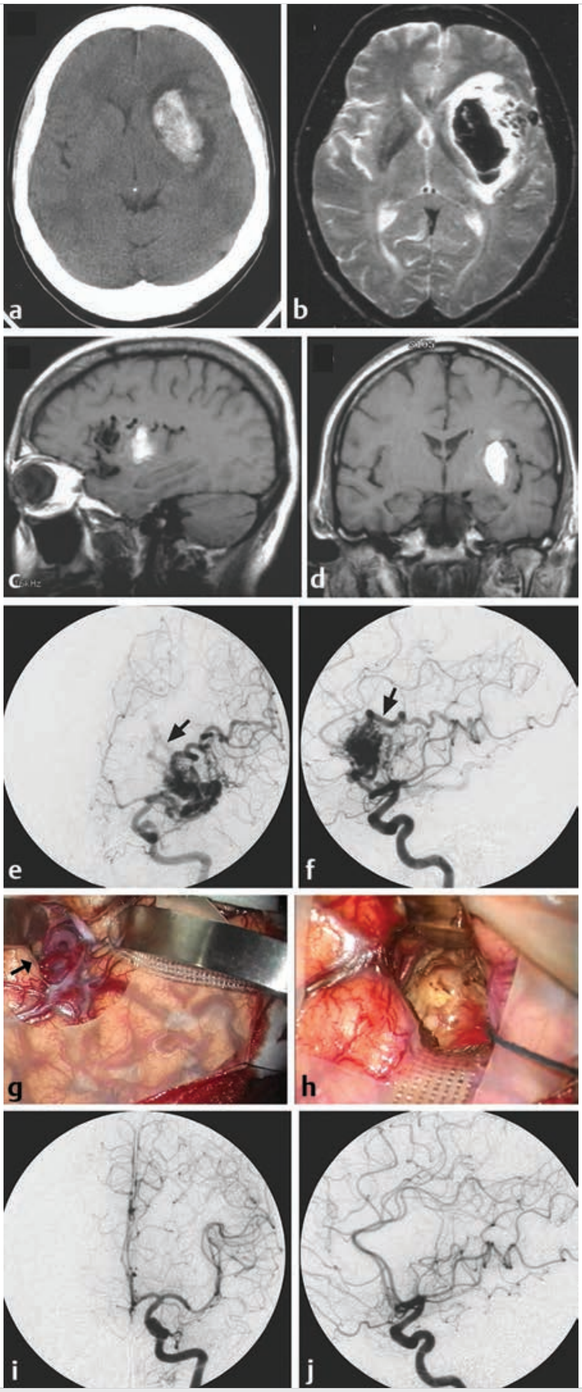

Fig. 24.6 A 48-year-old female presented with right-sided weakness and word-finding problems. **(a)** The initial computed tomography (CT) scan demonstrates a left-basal ganglia hematoma. **(b)** The magnetic resonance imaging (MRI, T2 axial), **(c)** T1 sagittal, **(d)** T1 coronal images show the basal ganglia hemorrhage and flow voids deep to the sylvian fissure. **(e)** The angiogram shows a 2.2-cm arteriovenous malformation (AVM) fed by branches of the left middle cerebral artery with superficial venous drainage (*arrow*), right internal carotid artery anteroposterior and **(f)** lateral injections. A transsylvian approach was chosen. **(g)** Superficial view of AVM (*arrow*) after opening the sylvian fissure and **(h)** after AVM resection. **(i)** Postoperative right internal carotid anteroposterior and **(j)** lateral angiogram views demonstrating complete resection (**Video 24.2**).

basal veins of Rosenthal, and the cerebellomesencephalic veins join to form the vein of Galen. These veins need to be gently separated to gain access to the pulvinar. The disadvantage of the supracerebellar infratentorial approach as compared with the occipital transtentorial approach is the need to separate the venous complex. Although the occipital transtentorial approach has the advantage of exposure above the vein of Galen, it allows access only to the midline and ipsilateral half of the cerebellomesencephalic fissure. Image guidance is used to define the shortest distance to the AVM. Aforementioned microsurgical techniques are used to resect the AVM.

24.3.6 Closure Techniques

Intraoperative angiography may be used to confirm AVM obliteration. If residual AVM is visualized, the surgeon will have to decide between further resection at that time, staged surgical resection at a later time, subsequent embolization, and radiosurgery. The benefit of further resection and cure must be carefully weighed against operative risk.

Meticulous hemostasis is critical. If intraoperative bleeding was minimal, we generally increase the blood pressure to an MAP of 90 mm Hg at the end of the case to ensure that the field is dry. However, if intraoperative bleeding was excessive, we raise the MAP to 75 to 80 mm Hg for testing hemostasis. We line the surgical bed with oxidized cellulose (Surgicel, Ethicon, Inc., Somerville, NJ) to promote hemostasis of small vessels. The dura is closed primarily or with the aid of a dural substitute. We use 4–0 braided nylon suture. The bone is replaced with titanium plates and screws, the galea is closed with 2–0 absorbable sutures, and staples are placed in the skin.

24.4 Postoperative Management Including Possible Complications

All patients should be monitored for 24 hours in an intensive care unit. Tight blood pressure parameters are established (usually MAP of 65–75 mm Hg if good hemostasis was obtained at the end of the case and 60–65 mm Hg if hemostasis was difficult or there was evidence of hyperperfusion syndrome or brain swelling). On the second postoperative day, we relax the blood pressure parameters to a range of 75 to 90 mm Hg. The patient is allowed to run normotensive pressures and is transferred to the floor if the postoperative course is uneventful by the third postoperative day.

Postoperative anticonvulsants are indicated for patients who have seizures preoperatively. Unless the case was done in a hybrid angiography-operative suite, we perform a catheter angiogram during the postoperative stay even if the patient had an intraoperative C-arm angiogram that suggested complete AVM obliteration.

Recurrent hemorrhage is the greatest risk for AVM patients; we cannot overemphasize the importance of meticulous hemostasis during closure and blood pressure control during the perioperative period. Other surgical complications to avoid include tissue damage secondary to retraction or manipulation,

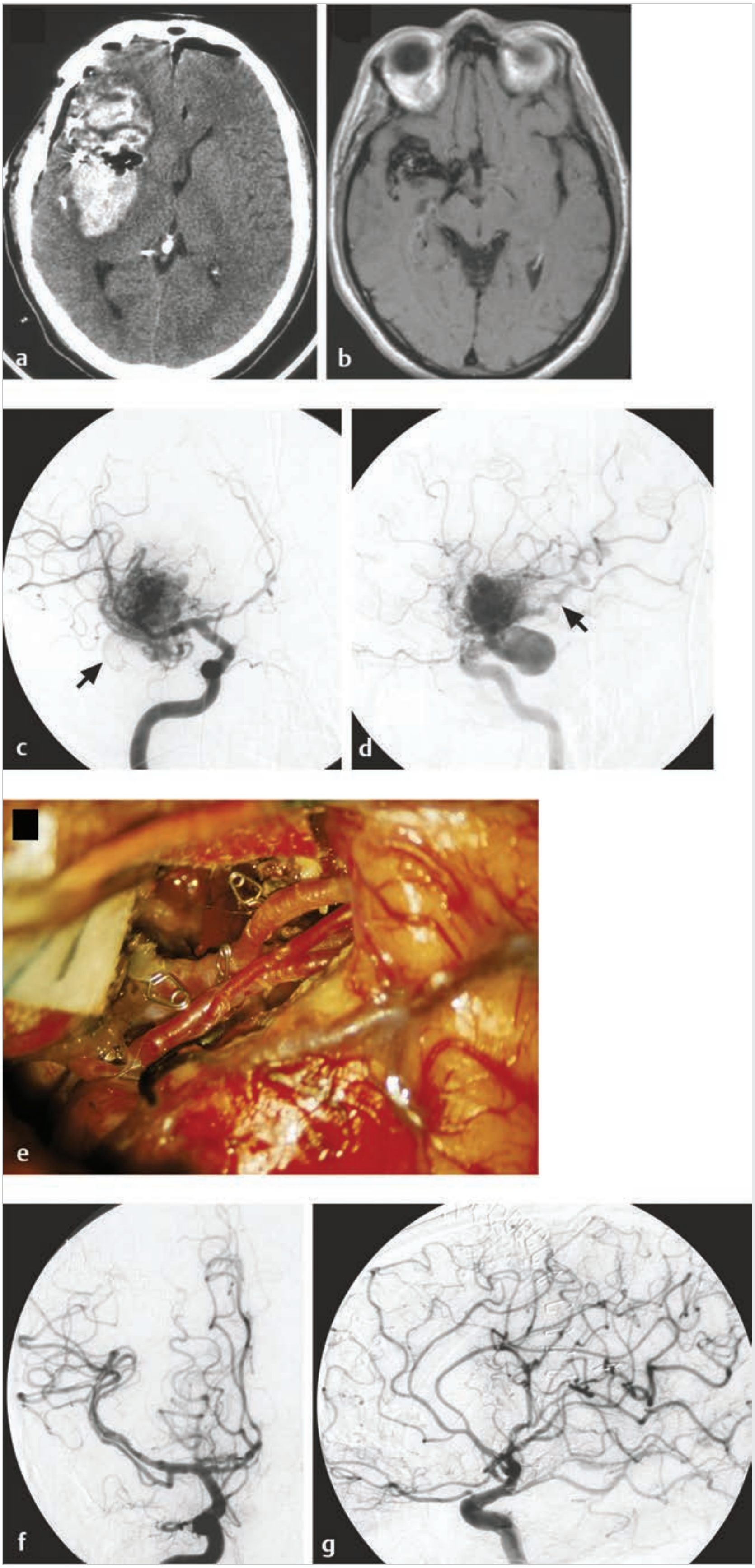

Fig. 24.7 A 53-year-old male presented with left hemiparesis. **(a)** The computed tomography (CT) scan shows a large, right-sided basal ganglia hemorrhage. **(b)** The arteriovenous malformation (AVM) is demonstrated on the magnetic resonance imaging (MRI) (T1 axial) extending from the basal ganglia to the insular cortex deep to the sylvian fissure. **(c)** On the right internal carotid angiogram, the AVM is fed by right lenticulostriates and **(d)** middle cerebral artery branches. The AVM has deep drainage **(c, d**, *arrows***)**. A transsylvian approach was used to resect the AVM. **(e)** Meticulous dissection in the sylvian fissure was required to differentiate normal middle cerebral artery branches and AVM feeding arteries originating directly from the middle cerebral artery branches. Feeding arteries were occluded using Sundt micro-AVM clips (Codman, Raynham, MA). **(f)** Postoperative anteroposterior and **(g)** lateral angiograms demonstrate complete resection.

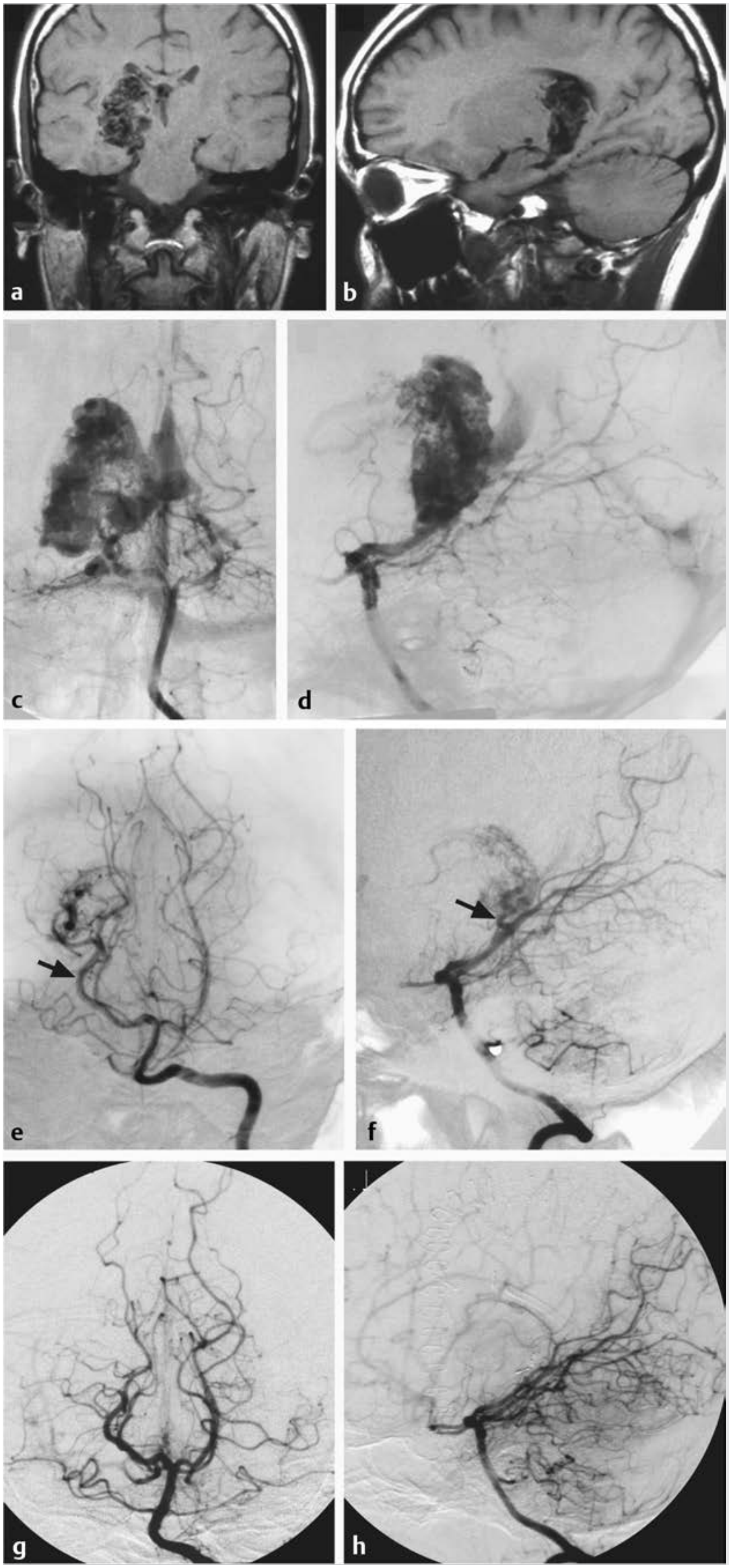

Fig. 24.8 A 22-year-old female presented with an acute onset of left homonymous hemianopsia and sensory deficit in her left lower extremity. **(a)** T1 coronal and **(b)** T1 sagittal magnetic resonance imaging revealed a right thalamic hemorrhage secondary to an arteriovenous malformation (AVM) in that location. **(c)** Anteroposterior and **(d)** lateral angiograms demonstrated a 3-cm AVM in the right thalamus fed by numerous small perforators arising from the anterior and posterior circulation. The vertebral artery injection is shown with deep venous drainage. The patient was treated with helium-ion radiosurgery reducing the lesion size by ~80% as shown on postradiosurgery anteroposterior **(e)** and lateral **(f)** vertebral artery angiograms, *arrow* shows predominant posterior cerebral artery supply. A right posterior temporal transcortical approach was performed to access the residual AVM. Ultrasound was used to localize the lesion deep to the cortex. The postoperative anteroposterior **(g)** and lateral **(h)** vertebral artery angiograms confirmed complete resection of the AVM.

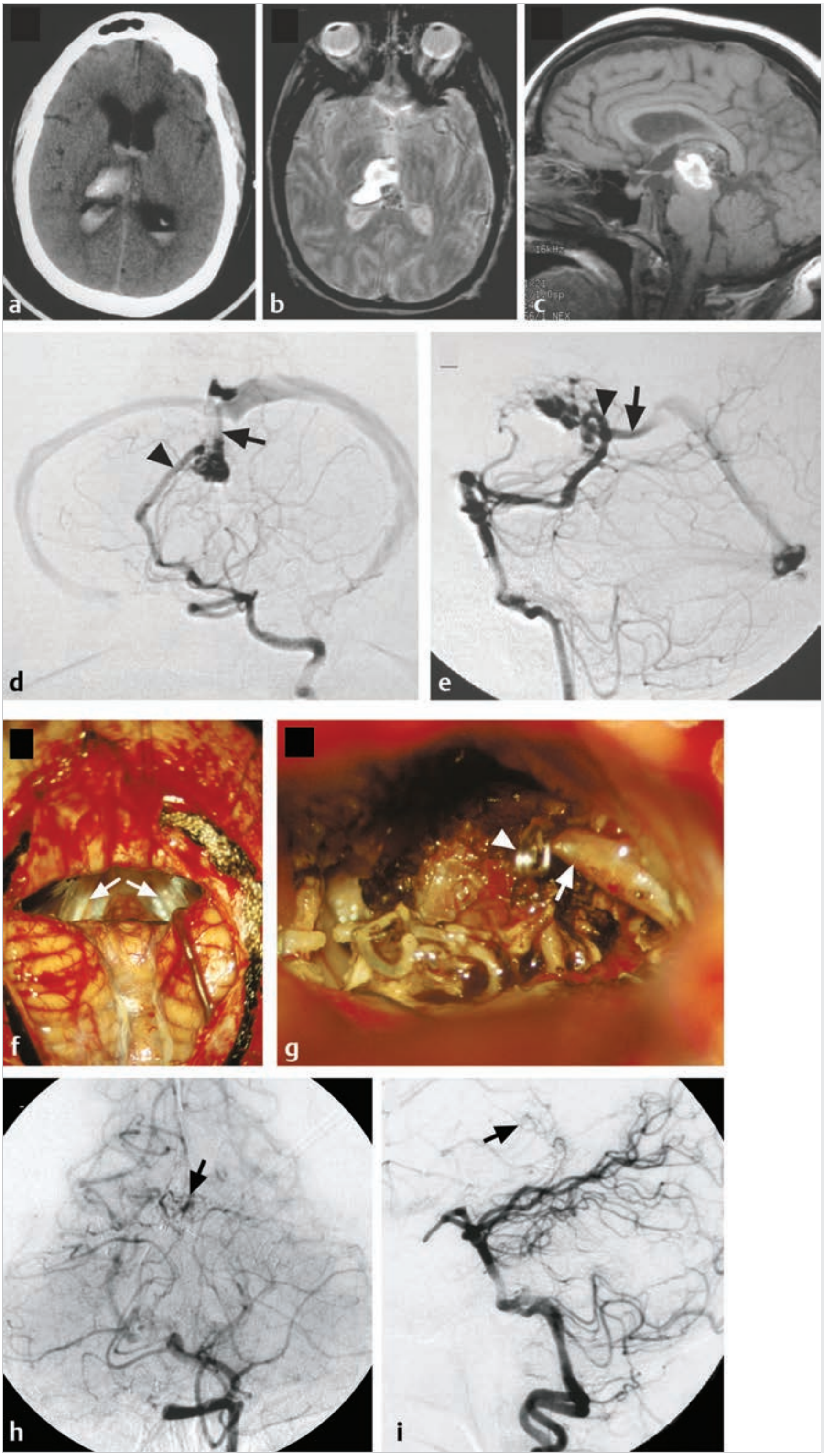

Fig. 24.9 A 44-year-old female initially presented with a loss of consciousness and coma. **(a–c)** Computed tomography scan **(a)** and axial T2 **(b)** and sagittal T1 **(c)** magnetic resonance imaging demonstrated a thalamic and intraventricular hemorrhage. She recovered from her initial hemorrhage apart from persistent left hemiparesis. The angiogram [**(d)** anteroposterior and **(e)** lateral vertebral injections] demonstrated a 2-cm arteriovenous malformation (AVM) fed by branches of the posterior cerebral artery (*arrowhead*) with deep venous drainage into the vein of Galen and straight sinus (*arrow*). A supracerebellar infratentorial approach in the semi-sitting position was performed giving access to both sides of the midline. **(f)** After a **Y**-shaped opening of the dura, both cerebellar hemispheres fall with gravity, opening the exposure to the quadrigeminal cistern. The internal cerebral veins, basal vein, and vein of the cerebellomesencephalic fissure can be appreciated (*arrows*). Image guidance was used to localize the AVM and guide resection. **(g)** The large feeding artery (*arrow*) is demonstrated and clipped (*arrowhead*). A tiny residual of the AVM was seen on the postoperative anteroposterior **(h)** and lateral **(i)** vertebral artery angiogram (*arrow*), was resected at a second surgery, and cured.

which may result in hemiparesis, hemiplegia, visual field defects, and aphasia. Overall, patients with AVMs in the basal ganglia or thalamus treated with surgery alone have a 20% worsening of motor function. Patients treated with embolization alone have a 40% risk of morbidity and 20% mortality. With stereotactic radiosurgery, patients with a preradiation surgical risk of 22% morbidity and 22% mortality improved to a postradiation surgical risk of 10% morbidity and 1.5% mortality.

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25 Intraventricular and Deep Arteriovenous Malformations

Michael Morgan and Nirav J. Patel

Abstract

The morphological and physiological complexity of brain arteriovenous malformations (AVMs) contributes to the challenge of management. When confronted with deeply located AVMs, involving periventricular, ventricular, insular, and basal ganglia locations, management decisions are compounded by access that often cannot be gained without a brain incision. The advent of advanced functional imaging, anesthetic considerations, microsurgical techniques specific for AVM surgery, and operative equipment has enabled surgical management of previously untreatable AVMs, with significant improvement in morbidity and mortality.

Keywords: arteriovenous malformation, ventricular, basal ganglia, thalamus, intracranial hemorrhage

25.1 Patient Selection

Choosing observation, microsurgical resection, focused irradiation, embolization, or some combination of these as the recommended management for deeply located arteriovenous malformations (AVMs) requires understanding of the risks and benefits of each of these pathways as well as of the natural history of the AVM. The risk for hemorrhage is 1 to 3% for unruptured AVM and 4 to 6% for AVM presenting with recent rupture.[1,2] Associated aneurysms and deep venous drainage system may also increase the hemorrhage risk for untreated unruptured AVM. Focused irradiation offers the benefit of avoiding the complex surgery necessary for deeply located AVMs but needs to take into account the latency period between treatment and obliteration during which time the natural history of rupture prevails.

Surgery has a high immediate cure rate but carries substantial risk for deep AVMs. Surgical risk depends on the size, location, and compactness of the nidus.[3,4,5,6] Most deep AVMs recommended for surgery are either Spetzler–Ponce class A or B.[6] There is a major difference in the risk of surgery between these two classes. Class A have a risk of permanent new neurological deficit less than 5% whereas it may be 10 to 24% for class B cases.[6,7,8] The risk benefit for class C may favor nonoperative management (▶ Fig. 25.1).

Embolization of the AVM as a single modality, or as a precursor to surgery or focused irradiation, is highly nuanced.[9] The authors of this chapter describe surgery without preoperative embolization.

25.2 Preoperative Preparation

25.2.1 Imaging

Catheter angiography with three-dimensional reconstructions and selective catheterization of feeding arteries, when additional information is required, are the gold standard for the evaluation and planning for surgical resection. Angiography accurately identifies arterial feeders and their location, diameter, length, and tortuosity as well as intranidal or arterial feeding aneurysms (▶ Fig. 25.2, ▶ Fig. 25.3). This information enables an estimate of the natural history of the lesion, as well as helps to prepare the surgeon for where feeders will be found during surgery.

Magnetic resonance imaging (MRI), potentially functional and diffusion tensor imaging, allows a better appreciation of the relation of the nidus to the surrounding brain and are useful for calculating AVM size, the relationship to the ventricles, sulci surface presentation of the lesion, and location of eloquent cortex and white matter (▶ Fig. 25.2).

Functional brain rarely exists within the interstices of the AVM. Therefore, if a marginal resection is achieved, it can be assumed, in most cases, that critically functioning brain can be preserved. Neurological deficits may develop in rare cases where functional brain exists within the AVM but more commonly

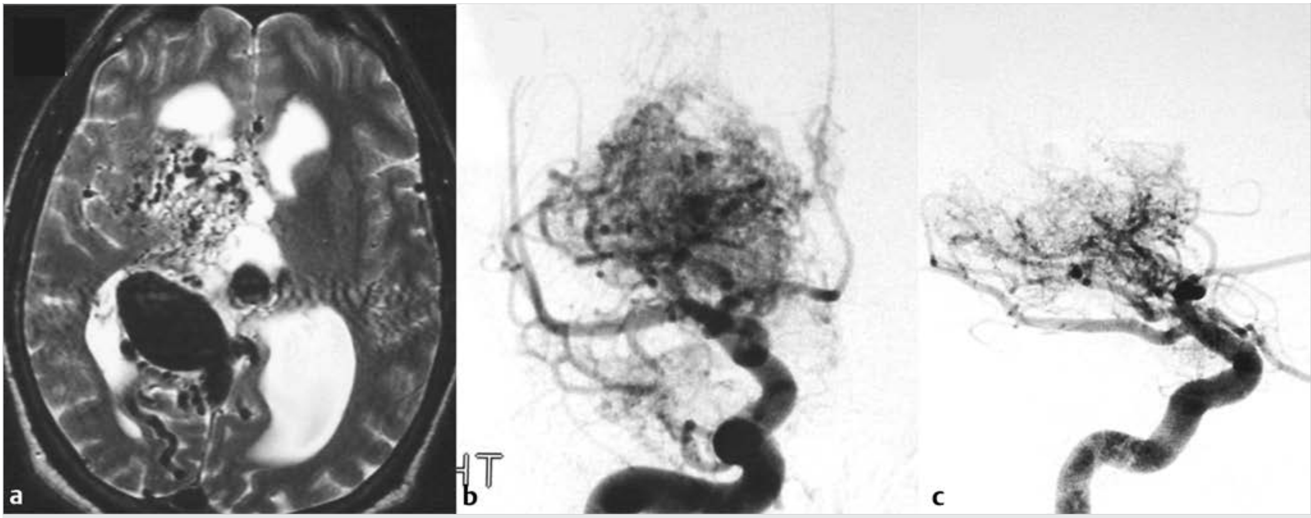

Fig. 25.1 Arteriovenous malformations (AVMs) chosen not to operate on by the authors. **(a)** Right hemispheric Spetzler–Martin grade 5 AVM. **(b)** Anteroposterior and **(c)** lateral digital subtraction angiogram of a right basal ganglia grade 5 AVM with lenticulostriate involvement.

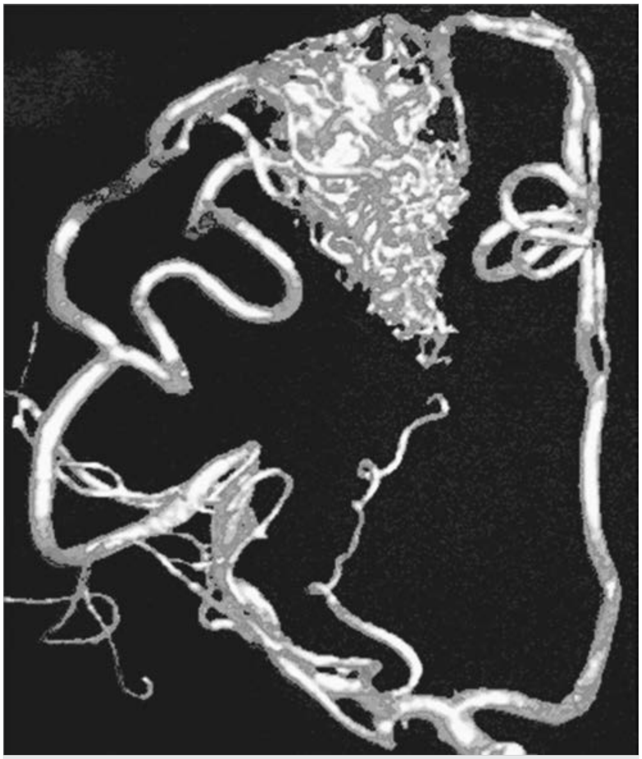

Fig. 25.2 Magnetic resonance imaging and catheter angiography of an arteriovenous malformation approached best through the floor of the left lateral ventricle.

when an artery "en passage" is occluded along with the AVM or the brain adjacent to the AVM is injured, usually in attempts to control bleeding from deep feeding arteries.

25.3 Operative Procedure

General principles for removal of deep AVMs are:

1. Craniotomies need to provide optimum exposure of the AVM in order to minimize retraction and maximize the ability to identify and access proximal arteries and venous drainage. For deep locations, consideration needs to be made of optimizing access through a fissure and sulcus. Insular AVMs require an exposure that allows the entire sylvian fissure to be opened. Medial frontal, parietal and occipital AVMs can be maximally exposed by operating on either side of the superior sagittal sinus with the contralateral approach through the falx cerebri for the most medial and the most lateral aspect of the AVM. Intermediately located AVMs may be more readily accessed by the ipsilateral approach. In this case, having the superior sagittal sinus perpendicular to the ground facilitates exposure. For lateral ventricular AVM, approaching the relevant lateral ventricle through a contralateral side assists in accessing the most lateral aspect of the AVM (**Video 25.1**).
2. Feeding arteries need to be dissected proximal as well as distal to all branches to the AVM. Preserving the distal arterial supply to normal brain is best achieved proceeding from distal to proximal, ligating and dividing terminal arteries supplying the AVM after determining the distal normal arterial supply. This strategy minimizes the potential inadvertent mistake of removing the normal distal artery (**Video 25.2,** ▶ Fig. 25.3).
3. Temporary arterial clips applied to feeders at a distance from the AVM provide regional hypotension within the AVM, provided that almost all feeding arteries are controlled (ensuring that the venous drainage is not impeded). This strategy improves the safety of dissection on the AVM margin.
4. Retraction should be minimized but is unlikely to be avoided in deeply placed AVM. Retraction of the brain may exacerbate ischemia. Light and limited retraction of the AVM is safe providing that venous outflow is not impeded. Impeding the venous outflow will increase the tension within the AVM.
5. Securing arteries by bipolar is made difficult by the thin walls of small feeding arteries (relative to the vessel radius). This makes coagulation difficult with bursting of the vessels if not performed carefully with clean bipolar tips at coagulation settings lower than normally employed. In addition, the use of microclips (e.g., as invented by Sundt) to arrest flow can assist either with diathermy or as the principle ligating agent.[10] Some surgeons prefer irrigating bipolars or disposable bipolar forceps. An additional technique suitable for some vessels includes incorporating adjacent brain with broad bipolar blades to spread the current and incorporate more material to assist with sealing vessels (**Video 25.3**).[11]
6. Securing all arterial input before venous drainage is compromised is critical. In order to achieve this without causing the most distal AVM becoming tense and easily ruptured is assisted by first securing the accessible superficial feeding arteries followed by a partial and limited AVM marginal arc dissection to the deepest component of the AVM (including entry into the ventricles if the AVM juxtaposes the ventricle) to secure arteries to the deep nose cone. This differs from the normal planned circumferential spiral, in a corkscrew approach, achieving an even depth of dissection on the margin of the lesion. This corkscrew circumferential dissection is not recommended until after the deep feeders are controlled. This is because the venous component of the AVM is located on the surface of the AVM and in the process of corkscrewing the dissection, this superficial venous drainage, internal to the AVM, may be increasingly compromised with preservation of a deep arterial contribution to the nose cone. Therefore, securing the deep feeding arterial supply should precede a circumferentially deepening dissection of the resection margin (**Video 25.4**).
7. Small arterialized vessels must be followed for a short distance into the white matter during the corkscrew circumferential dissection before a decision to ligate these vessels is made. This is because it is not easily possible to distinguish small feeding arteries from arterialized venous loops that leave and then rejoin the AVM. The cumulative ligation of venous loops will progressively impede venous drainage from the depth of the AVM. The final nose cone then may become very tense if its arterial input has not been secured. Therefore, the venous loops should be left unligated and only the infrequent arterial input ligated and divided.
8. Before ligation and division of the main draining vein is attempted, the resected AVM should be delivered from the resection bed with the small umbilical attachment of the draining vein remaining.

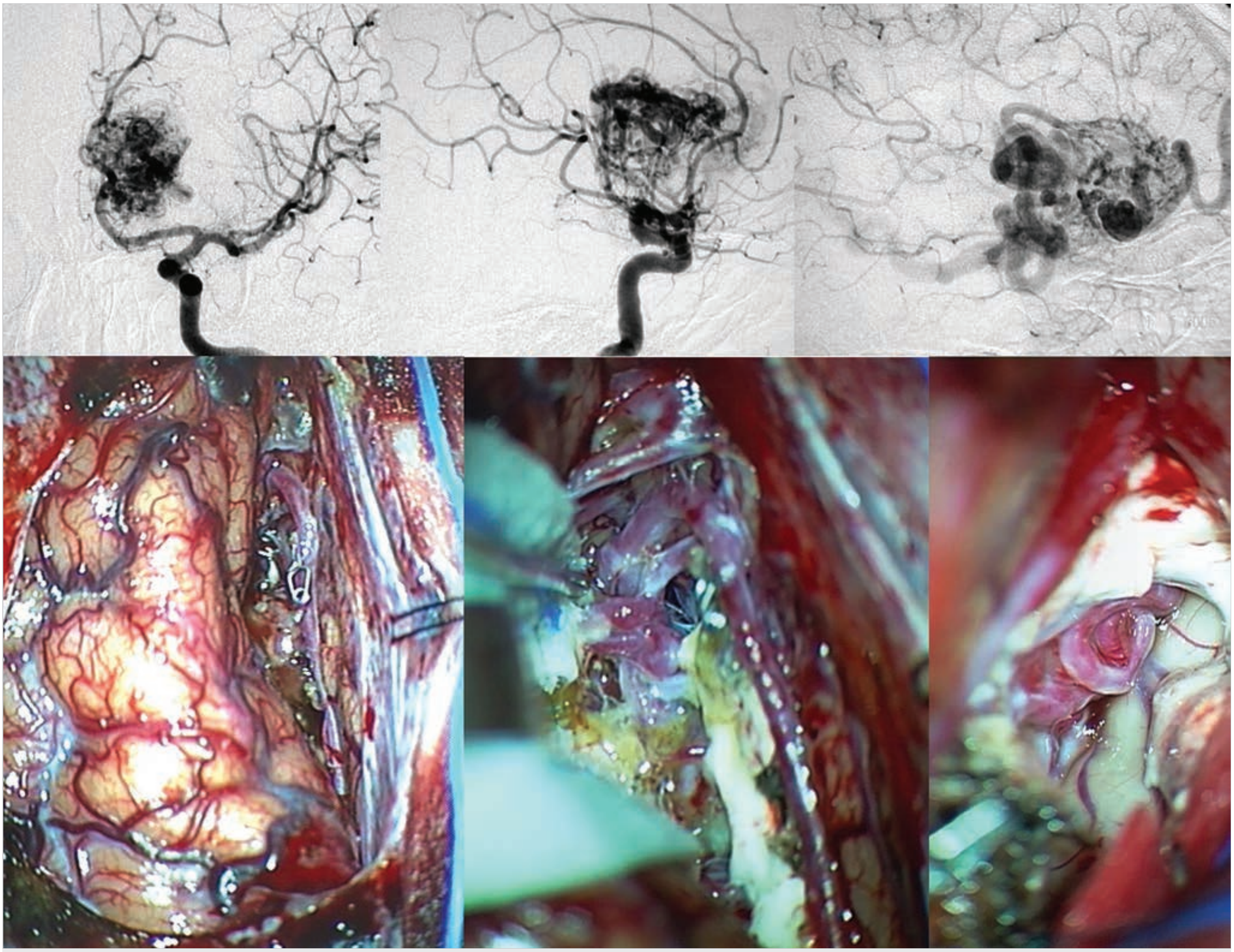

Fig. 25.3 Catheter angiography of an arteriovenous malformation (AVM) of the corpus callosum, caudate, hypothalamus, and third ventricle. This was approached through an anterior transcallosal incision having exposed a long segment of the anterior cerebral artery before callosotomy and AVM resection.

9. Inspection of the resection bed should find absolutely no arterialized bleeding. Any bleeding should raise the possibility of retained AVM that may lead to catastrophic postoperative hemorrhage. It is important not to stress test the security of hemostasis by raising blood pressure during the surgery. Any area in the bed of the AVM with arterialized bleeding must be explored. Irrespective of whether this bleeding is due to retained AVM or not, the small vessels responsible for persistent bleeding have not the capacity to constrict due to their minimal muscular wall. Bleeding needs to be specifically surgically arrested rather than allow time to pass for and anticipated arterial vasoconstriction response for control.
10. In cases where there is meningeal supply to the AVM, it is important that the dura and brain not be retracted apart as the bridging artery may tear. An indirect approach to these feeding arteries within the dura is appropriate by creating an island of dura and leaving this on the brain over the AVM. The tentorium cerebelli or the falx cerebri can be incised at a considerable distance from the AVM securing the blood supply (e.g., division of the tentorium from lateral to medial will secure the meningeal arterial supply from the carotid artery or opening of the falx from a contralateral approach at a distance from the nidus can deal with feeders entering from the falx).

Another important consideration is the impact of the dissection through the brain when obtaining access to the deep feeders. Thought needs to be given not only to the function of the tissue to be divided (such as the association fibers) but also to whether tandem lesions will be created. Two tandem lesions that need consideration include whether the dominant visual cortex is damaged if the splenium is intended to be divided, because this would render the language cortex to be disconnected from functional visual cortex. The other is whether previous external ventricular drain insertion may have injured one or the other of the fornices when one is planning to enter contralateral memory systems such as the fornix, hypothalamus, and thalamus. Inadvertent bilateral injury may have catastrophic effects upon memory function.

One final point concerns previously inoperable AVMs in eloquent areas that are selected for surgical intervention. In cases of massive hemorrhage or infarction that induce permanent focal neurological deficit or are life threatening, direct surgical corridors through overlying eloquent cortex may be indicated and be feasible without creating additional neurological deficits (▶ Fig. 25.4).

25.4 Specific Approaches to Deep AVM by Location

25.4.1 Anterior Interhemispheric Approach

AVMs accessed by this approach are often fed by both anterior and middle cerebral arteries (▶ Fig. 25.6). Supply is frequently from the recurrent artery of Heubner, posterior perforating branches of the anterior communicating artery, inferior branches of the callosal-marginal and pericallosal and medial branches of the lenticulostriate arteries. There may be arterial supply from anterior choroidal branches, but these are usually small in comparison. If the AVM is predominantly located at the anterior corpus callosal region, the venous drainage is usually into the sagittal sinus and intraventricularly to the septal, thalamostriate, and, subsequently, the internal cerebral veins.

AVMs within this location can be approached in one of two ways: interhemispheric transcallosal (▶ Fig. 25.6**, Video 25.5**) or transcortical transventricular. We prefer the interhemispheric approach for lesions within or immediately adjacent to the ventricle because it allows the ability to follow and skeletonize the branches of the callosomarginal and pericallosal proximally, while allowing good marginal exposure of the AVM. The exposure allows adequate retraction in sagittal and coronal planes, providing intraventricular and transependymal access enabling arterial feeder exposure, and is best used in the case of small ventricles. The transcortical approach may be preferred for deep paraventricular lesions with a minimal intraventricular component or with intraventricular components in the case of large ventricles.

It is essential to analyze the preoperative angiogram and MRI to determine if laterally placed AVMs recruit the lateral branches of the lenticulostriate feeders. This greatly increases the surgical risk due to internal capsular involvement, with some authors concluding that lateral lenticulostriate involvement renders the AVM inoperable due to the high risk of surgical morbidity. Also, the further the AVM extends away from the midline the harder it is to approach from the midline interhemispheric approach.

The patient is placed supine in a neutral position or with ipsilateral rotation, with three-point pin fixation. The head is flexed slightly, avoiding venous compression, and the bed is positioned such that the patient's head is above the level of the heart to minimize venous pressures. A craniotomy is performed mostly anterior to the coronal suture, allowing exposure of the sagittal sinus. An interhemispheric approach is utilized, with appropriate retraction. The dura is opened and reflected against the superior sagittal sinus (**Video 25.4, 25.5,** ▶ Fig. 25.3). The medial aspect of the frontal lobe is retracted, ensuring the major draining vein is unimpeded. Arachnoid dissection is usually necessary to adequately identify the pericallosal and callosomarginal arteries along with the course of the draining vein adjacent to the cingulate gyrus. The pericallosal and callosomarginal arteries are identified, and if inferior branches are involved these are followed carefully and divided only when confirmed to be entering the AVM nidus.

We start the arterial dissection distally, beyond the last major contributor to the AVM nidus, and work proximally because this allows ready identification of the important arteries that pass by the AVM and need to be preserved. On the medial side, one divides the corpus callosum, taking care not to damage the fornix. One can secure anterior cerebral and choroidal arterial supply from this side. Marginal resection is then performed as already indicated in the section on general principles, with the use of low suction, bipolar coagulation and microclips, as necessary. Hemostasis is particularly important since any postoperative bleeding into the ventricles increases the risk of permanent hydrocephalus.

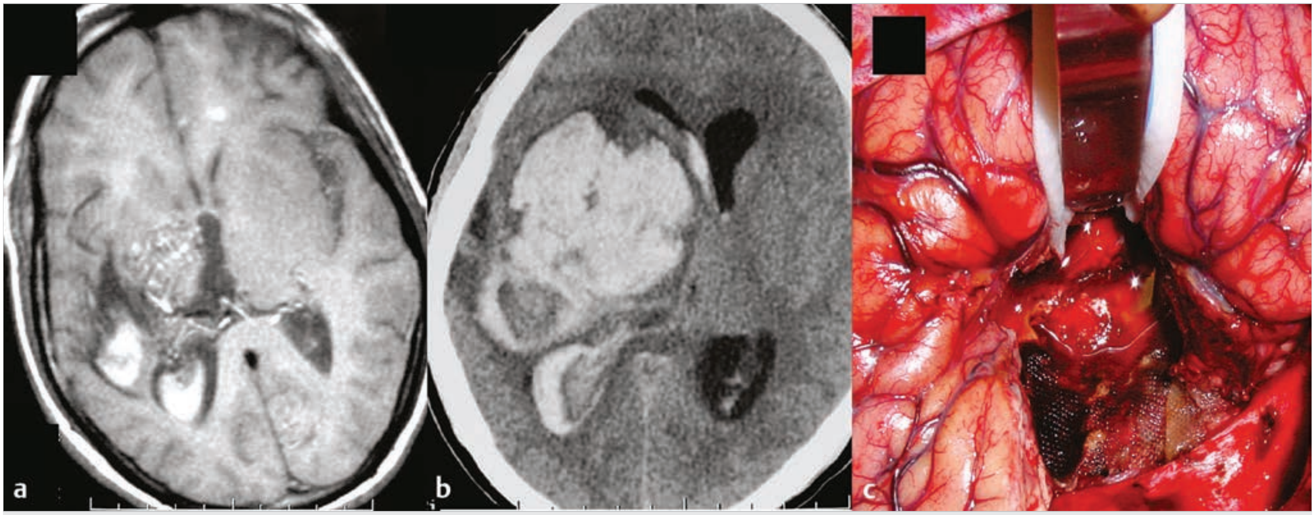

Fig. 25.4 **(a)** T1-weighted magnetic resonance imaging showing acute hemorrhage from a deep right arteriovenous malformation (AVM). **(b)** Computed tomography showing massive hemorrhage secondary to a deep right AVM. This mandated surgical extirpation. **(c)** The surgical corridor through the cortex overlying the AVM can be seen.

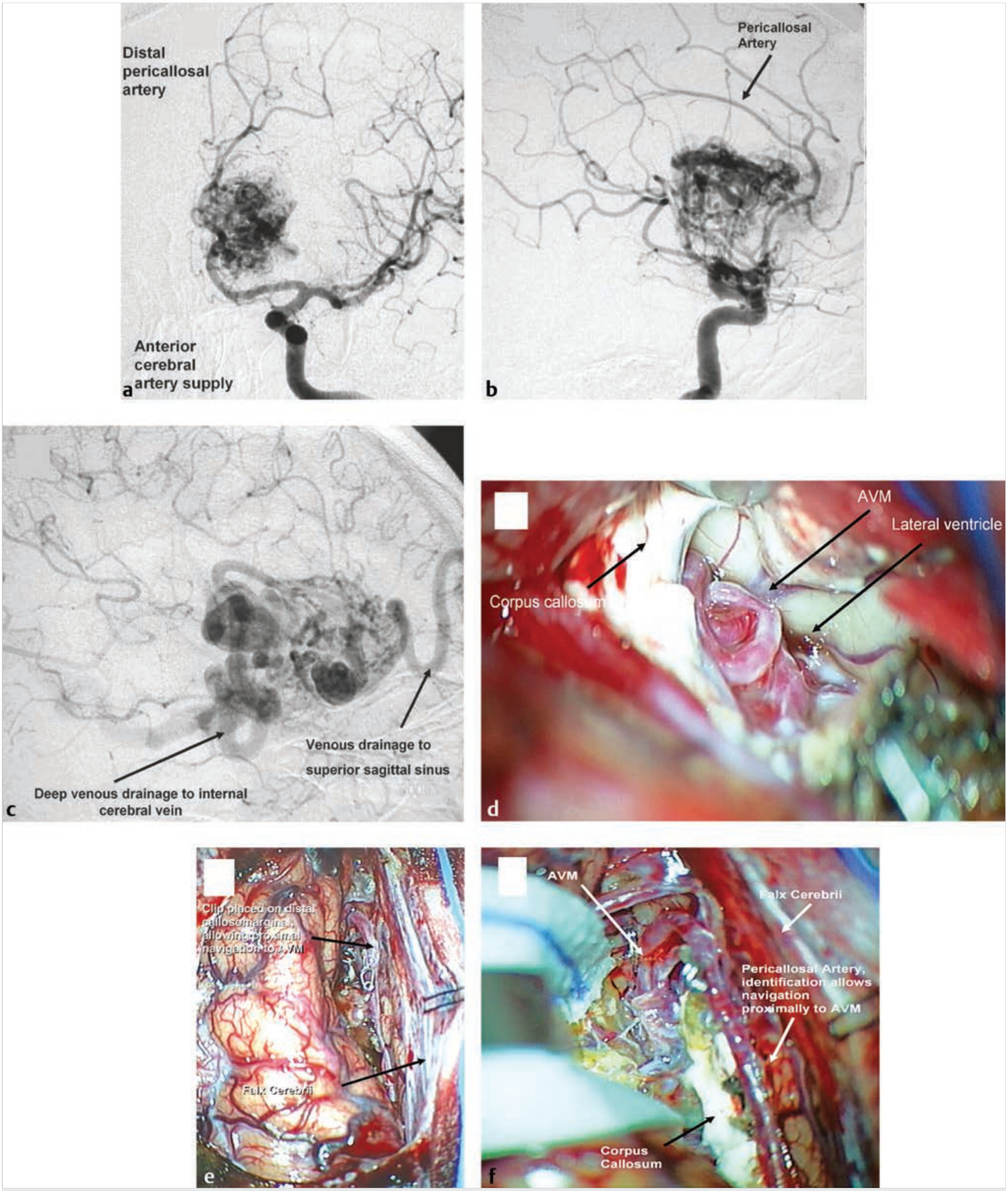
Distal pericallosal artery
Anterior cerebral artery supply
a
Pericallosal Artery
b
Deep venous drainage to internal cerebral vein
Venous drainage to superior sagittal sinus
c
AVM
Lateral ventricle
Corpus callosum
d
Clip placed on distal callosomarginal, allowing proximal navigation to AVM
Falx Cerebrii
e
AVM
Falx Cerebrii
Pericallosal Artery, identification allows navigation proximally to AVM
Corpus Callosum
f

Fig. 25.5 **(a)** Digital subtraction angiography showing anteroposterior and **(b)** lateral arterial phase and **(c)** lateral venous phase views of a third ventricular arteriovenous malformation (AVM). The lesion was resected through an anterior transcallosal approach **(d–f)**. **(d)** Intraoperative approach to the anterior third ventricular AVM. **(e)** Intraoperative interhemispheric approach to anterior third ventricular AVM showing skeletonization of the pericallosal artery. **(f)** Magnified view of the interhemispheric approach with identification of the pericallosal artery and AVM.

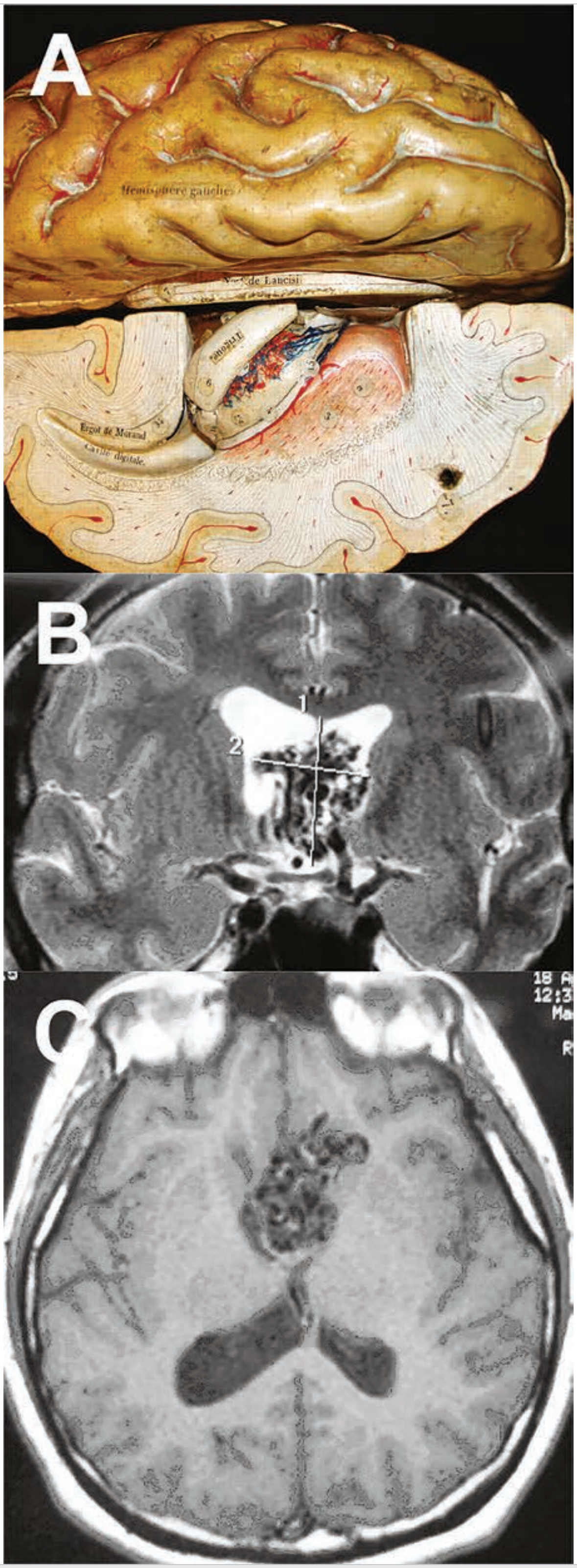

Fig. 25.6 **(a)** Model of brain highlighting relevant anatomy for the anterior transcallosal approach. **(b)** T2-weighted coronal magnetic resonance imaging showing an anterior third ventricular arteriovenous malformation (AVM). **(c)** Axial T1-weighted magnetic resonance image showing an anterior third ventricular AVM.

25.4.2 Posterior Interhemispheric Approach

AVMs of the posterior third ventricle and splenial regions are generally supplied by the posterior cerebral artery, the medial and lateral choroidal arteries, and the distal pericallosal branch of the anterior cerebral artery (▶ Fig. 25.7, ▶ Fig. 25.8). There may be additional supply from the distal middle cerebral artery branches, but this is unusual. Venous drainage is usually directly to the internal cerebral veins or vein of Galen into the straight sinus.

We prefer a direct interhemispheric or parasagittal approach, depending upon the laterality of the lesion. The patient is positioned prone, in three-point pin fixation, with the head flexed to the point that allows a direct vertical access to the splenium from a midline surface plane 3 cm forward from the posterior occipital protuberance. A craniotomy is performed crossing the sagittal suture to enable adequate exposure of the superior sagittal sinus. The splenium is divided, having carefully considered the potential for symptomatic disconnection syndromes to be unmasked, and the posterior third ventricle is accessible through the quadrigeminal cistern. The interhemispheric approach in this position enables early security of the pericallosal feeders before identification of the posterior choroidal artery arising through the quadrigeminal cistern.

It is essential to determine the margins of the AVM and avoid proximal arterial clipping of the choroidal arteries because this can cause infarction and neurological deficit. As with the anterior interhemispheric approach, difficulty may arise with lateral exposure, particularly when the AVM extends laterally into the trigone. Lateral visualization can be achieved by a contralateral approach (in addition to the side of the AVM) and dividing the falx to achieve a greater angle of trajectory (**Video 25.1**).

Posterior third ventricular approaches require special care to avoid damage to the internal cerebral veins. A further option for lateral exposure is division of the cingulate gyrus. Again, consideration needs to be taken of previous or synchronous tandem brain lesions, and the general principles of deep AVM surgery discussed earlier should be followed.

25.4.3 Sylvian Approach to Insular AVM (Video 25.6)

This approach is similar to a large sylvian approach for aneurysms. The head is only slightly turned to the opposite side with the head slightly extended and raised above the level of the heart. The aim is to have the sylvian fissure perpendicular to the ground. This facilitates sylvian fissure opening and minimizes retraction of either temporal or frontal lobe.

The middle cerebral veins will likely be arterialized and it is critical not to injure these during the dissection. Sharp dissection and wide opening of the sylvian fissure is appropriate to ensure that the middle cerebral artery, both proximal and distal to the AVM dedicated branches, can be readily identified and protected. Although the arterialized superficial middle cerebral and deep middle cerebral vein may increase the difficulties in the wide opening of the sylvian fissure, the dilated feeding arteries that have often lengthened within the fissure generally make the sylvian fissure widely opened once entered.

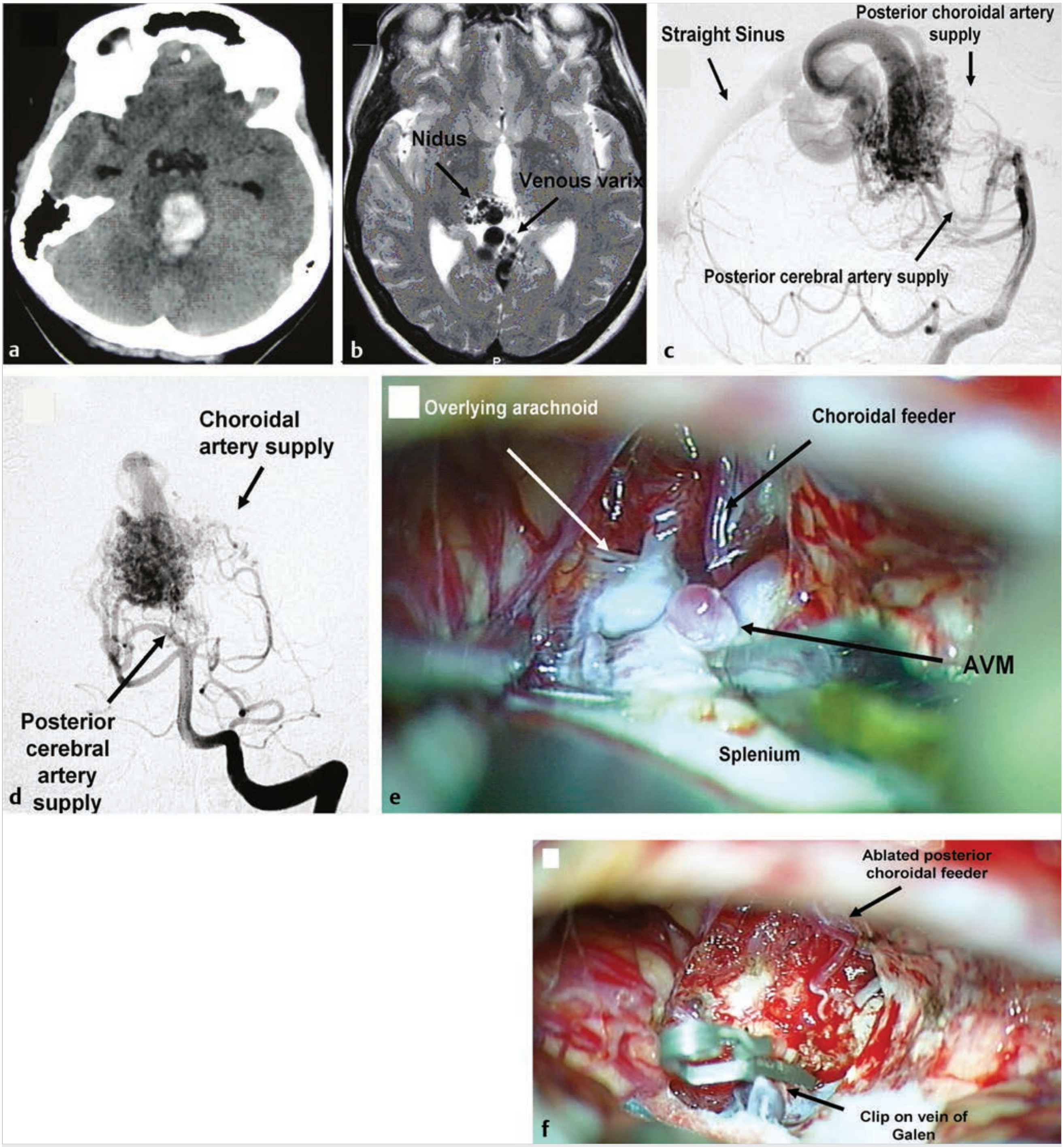
a
Nidus
Venous varix
b
Straight Sinus
Posterior choroidal artery supply
Posterior cerebral artery supply
c
Choroidal artery supply
Posterior cerebral artery supply
d
Overlying arachnoid
Choroidal feeder
AVM
Splenium
e
Ablated posterior choroidal feeder
Clip on vein of Galen
f

Fig. 25.7 **(a)** Axial computed tomographic scan showing acute hemorrhage in the posterior callosal region. **(b)** T2-weighted axial magnetic resonance imaging showing a posterior callosal arteriovenous malformation (AVM) with nidus and venous varix. Digital subtraction angiograms with **(c)** lateral and **(d)** anteroposterior views showing the posterior callosal AVM with arterial supply from the posterior cerebral and posterior choroidal arteries and marked venous engorgement. **(e)** Intraoperative exposure of the AVM with the arachnoid being dissected with a number 11 blade knife. **(f)** Removal of the posterior callosal AVM showing ablated posterior choroidal feeder.

Dissection should proceed from distal to proximal. This ensures that during the arterial feeder ligation the distal flow to the brain is secure. The feeding arteries to the AVM are often identified by their large size (for location) and their slight recurrent course. The corticotomy must be on the margin of the AVM in this insula location.

25.5 Postoperative Management Including Possible Complications

All patients are transferred to neurointensive care, with most Spetzler–Ponce class B (or more complex) AVM cases kept

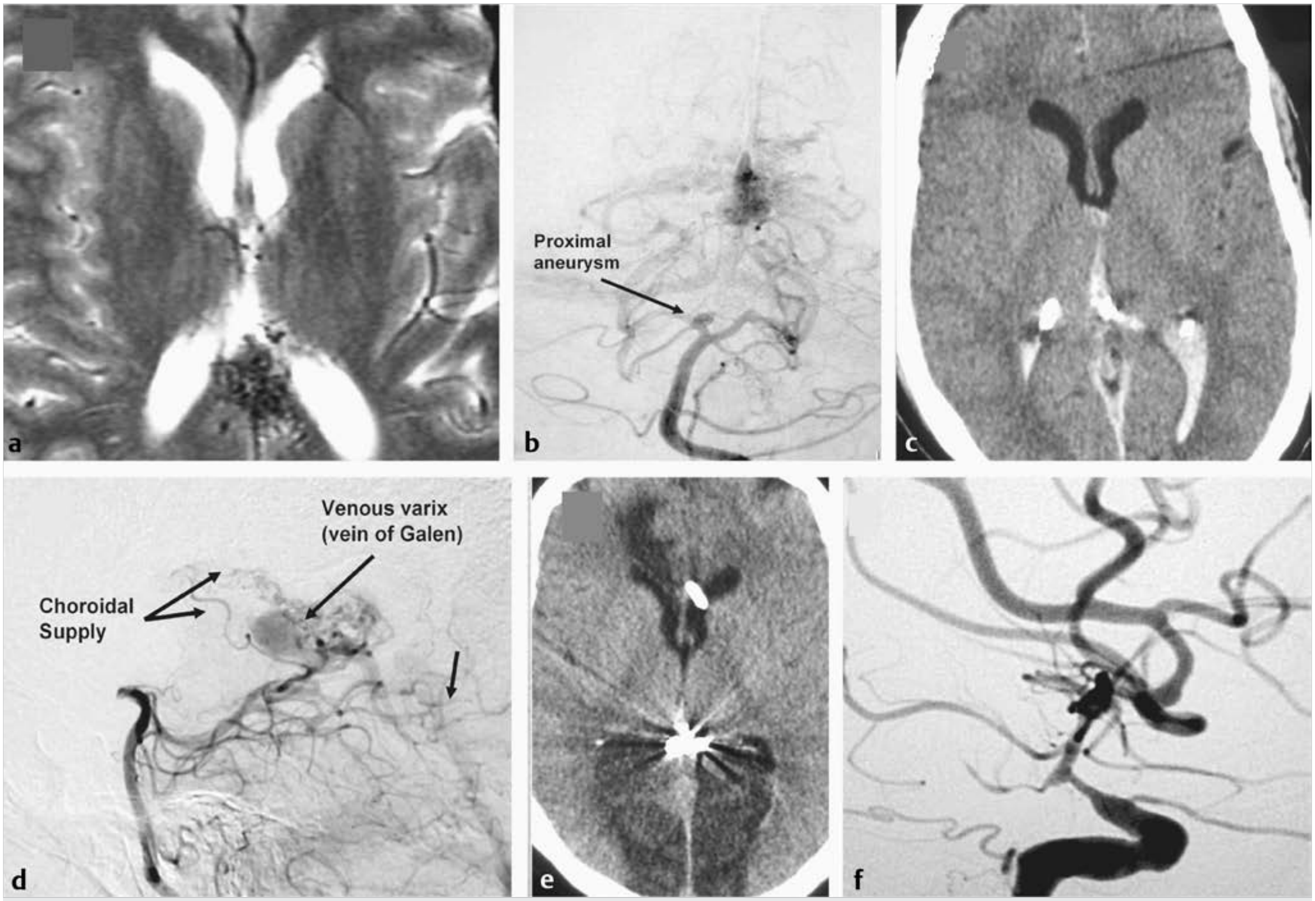

Fig. 25.8 (a) T2-weighted axial magnetic resonance imaging showing posterior location of a splenial arteriovenous malformation (AVM). **(b)** Anteroposterior vertebral digital subtraction angiogram showing arterial supply to the AVM and identification of a proximal aneurysm. **(c)** Axial computed tomography (CT) shows hemorrhage from the splenial AVM. **(d)** Lateral vertebral angiogram showing arterial feeders and venous drainage. **(e)** Axial CT 5 days after removal of the AVM shows bilateral occipital infarcts. Surgical extirpation was uncomplicated with the CT performed secondary to new-onset visual loss 5 days after extirpation. **(f)** Digital subtraction angiogram showing severe vasospasm following initial hemorrhage and surgical ablation of the AVM.

intubated and sedated. Class A patients can be allowed to emerge immediately from anesthesia. Strict mean arterial blood pressure control is instituted with larger class B and C cases, allowing a maximum pressure of 70 mm Hg with cerebral perfusion pressure aimed to be more than 50 mm Hg.[12] We obtain a computed tomographic (CT) scan and angiogram soon after surgery but delay the catheter angiogram until 6 or 7 days after surgery. Postoperative neurological deterioration mandates CT and usually some type of angiogram to detect postoperative hemorrhage.

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26 Vein of Galen Malformations

Jason A. Ellis, Nikita G. Alexiades, Randall T. Higashida, and Philip M. Meyers

Abstract

Vein of Galen aneurysmal malformations (VGAMs) are rare congenital cerebrovascular malformations that share the common feature of dilation of the vein of Galen or its embryologic precursor, the median prosencephalic vein. These lesions comprise 30 to 40% of all pediatric intracranial vascular malformations and have an incidence of less than 1%.[1,2] Endovascular methods are the mainstay of treatment. Nonetheless, surgical intervention may be used to facilitate definitive endovascular treatment or as an ancillary approach in the setting of hydrocephalus requiring cerebrospinal fluid diversion or hemorrhage necessitating evacuation.

Keywords: arteriovenous malformation, vein of Galen

26.1 Introduction

Vein of Galen aneurysmal malformations (VGAMs) are rare congenital cerebrovascular malformations that share the common feature of dilation of the vein of Galen or its embryologic precursor, the median prosencephalic vein (▶ Fig. 26.1 and ▶ Fig. 26.2). These lesions comprise 30 to 40% of all pediatric intracranial vascular malformations and have an incidence of less than 1%.[1,2]

VGAMs are distinct from anomalies such as vein of Galen aneurysmal dilation or vein of Galen varix that simply manifest with dilation of the true vein of Galen. VGAMs are characterized by arteriovenous shunting into a persistent median prosencephalic vein of Markowski, the embryologic precursor of the vein of Galen. This distinction between VGAM and vein of Galen aneurysmal dilation or varix is critical because there are implications for treatment. The median prosencephalic vein is an embryological remnant that continues to exist because of pathological arteriovenous shunting, and thus it can often be occluded without impairment of venous drainage in the surrounding brain. This is not generally true for the vein of Galen. The vein of Galen in a vein of Galen aneurysmal dilation or varix typically retains the essential role of draining the deep cerebral venous system and should not be occluded.

26.2 Patient Selection

26.2.1 Clinical Presentation

The presentation of patients with VGAMs is diverse and contingent upon lesion angioarchitecture, the presence of comorbidities and the age of the patient. Early, fulminant presentations suggest higher degrees of arteriovenous shunting and, as a result, portend more severe morbidity and increased mortality.[3,4] In neonates, the initial presentation is frequently congestive heart failure with cyanosis, respiratory distress, and electrocardiographic changes (▶ Fig. 26.3). Up to 95% of patients with antenatally diagnosed VGAM presented with systemic cardiac symptoms at birth. Severe pulmonary hypertension, systemic ischemia, and prenatal hydrops may be present. Such presentations may be further complicated by diastolic flow reversal in the descending aorta particularly in patients with right to left cardiac shunting due to a patent foramen ovale or ductus arteriosus.[5]

The presentation of VGAMs in infants and young children are usually with lesser degrees of arteriovenous shunting and thus

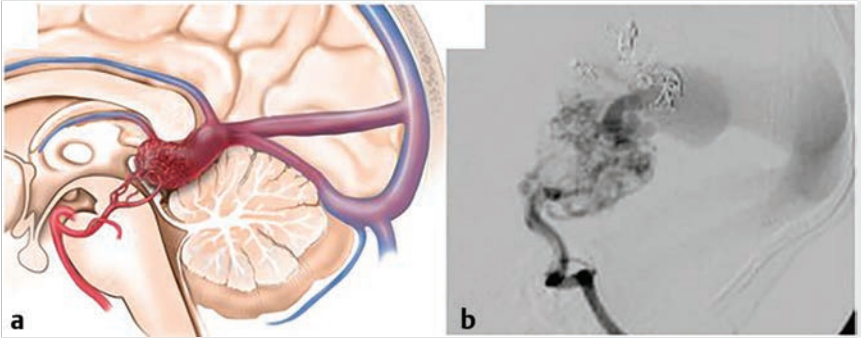

Fig. 26.1 A choroidal vein of Galen aneurysmal malformation demonstrating the anterior nidal network of arterial feeders is shown by artist depiction **(a)** and after vertebral angiography **(b).**

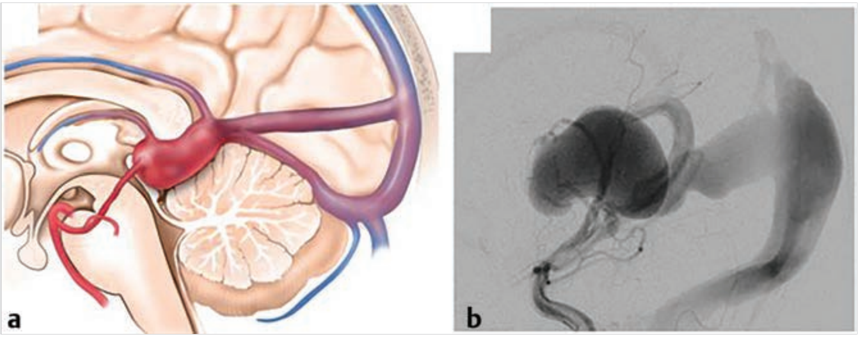

Fig. 26.2 A mural vein of Galen aneurysmal malformation demonstrating direct arteriovenous fistulas to the wall of the median prosencephalic vein is shown by artist **(a)** depiction and after angiography **(b).**

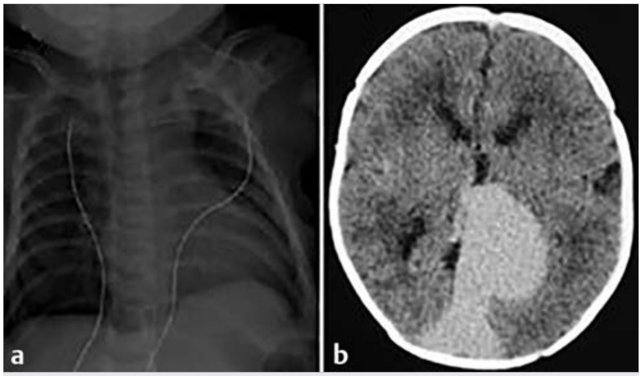

Fig. 26.3 This newborn presented with evidence of congestive heart failure. Chest X-ray demonstrates cardiomegaly and pulmonary congestion **(a)**. Noncontrast head computed tomography shows the presence of a vein of Galen aneurysmal malformation without significant hydrocephalus **(b)**.

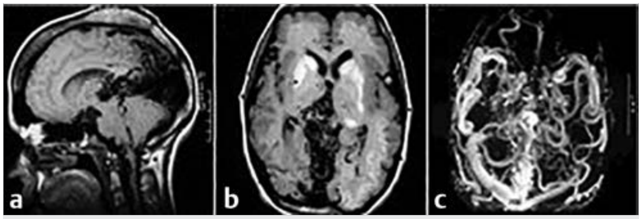

Fig. 26.5 This adult patient with a partially treated vein of Galen aneurysmal malformation during infancy presented 20 years later with epilepsy, hemiparesis, and dementia. Chronic brain parenchymal changes including atrophy, gliosis, and dysplastic vascular proliferation are evident on magnetic resonance imaging and angiography **(a–c)**. Interestingly, the patient showed dramatic cognitive improvement after further treatment and was able to find employment.

a more insidious clinical course. Hydrocephalus and increased head circumference, headaches, seizures, and developmental delay are commonly seen. Focal neurologic signs and symptoms or psychomotor delay due to vascular steal may also be seen in this age group.[5] Findings may include evidence of incipient heart failure, but more commonly, signs such as progressively increasing head circumference, dilated scalp and facial veins, and failure to achieve appropriate developmental milestones are noted.[4] Hydrocephalus may develop secondary to obstruction of the cerebral aqueduct, hemorrhage, venous sinus occlusion, or intracranial venous hypertension. Cranial bruits, proptosis, and recurrent epistaxis may also be observed in this population.[5]

26.2.2 Preoperative Evaluation

The angioarchitecture of VGAMs can be complex. Several classification schemes have been reported. An understanding of the detailed angioarchitecture of a VGAM under consideration for treatment is critical for determining the optimal therapeutic approach. The historically interesting VGAM classifications by Litvak and, later, Yasargil have been simplified by Lasjaunias.[6]

Lasjaunias classified VGAMs into choroidal and mural subtypes.[3,7,8] Choroidal VGAMs are characterized by arterial feeders from choroidal vessels forming a nidal network draining into the anterior aspect of the median prosencephalic vein (▶ Fig. 26.1). The multiple high-flow fistulas present in choroidal VGAMs typically result in early symptomatic expression during the neonatal period. Mural VGAMs are characterized by at least one direct arteriovenous fistula within the wall of the median prosencephalic vein (▶ Fig. 26.2).[8] The site of fistulous drainage is often at the inferolateral wall of this primitive vein. Mural VGAM tend to have lower flow through the fistulous connection and thus frequently present outside of the neonatal period with relatively milder but insidious symptoms.[9]

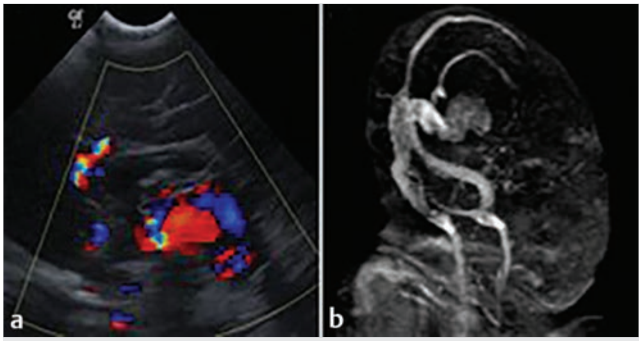

Fig. 26.4 Transfontanelle color Doppler ultrasound indicates the presence of a dilated midline venous collector in the region of the normal vein of Galen **(a)**. Subsequent magnetic resonance venography confirms the structure to be a vein of Galen aneurysmal malformation **(b)**.

Not all VGAMs fit nicely into either Lasjaunias choroidal or mural subtypes. Indeed, it is increasingly recognized that intermediate subtypes with features of both choroidal and mural VGAMs exist.[3]

Effective management of VGAMs necessitates a multidisciplinary approach involving pediatric medical, surgical, and interventional specialists. Emergent intervention is not always required, allowing time for preoperative optimization of cardiac and pulmonary status in many cases. However, some patients in high-output cardiac failure may need to be treated immediately. Diagnostic workup should begin with a physical examination including weight, head circumference, assessment of peripheral perfusion, neurological testing, and cardiopulmonary evaluation. Hepatic and renal function should be assessed particularly in the setting of cardiac insufficiency and systemic ischemia. An echocardiogram should be performed either antenatally when a prenatal diagnosis is made or on the first day of life. Documentation of baseline cardiac function and identification of associated congenital cardiac malformations is of critical importance in this patient population. Electrocardiography should be performed to identify evidence of myocardial ischemia and any conduction abnormalities.[9,10] When indicated, respiratory support should be initiated with supportive oxygen therapy via nasal cannula, CPAP/BiPAP, or intubation as appropriate. Digitalis derivatives and/or diuretic therapy may have utility in the setting of volume overload.[1,9]

Radiographic evaluation should include noninvasive modalities when possible. In neonates transfontanelle ultrasound can be performed rapidly at the bedside to evaluate for hemorrhage, hydrocephalus, and gross VGAM characterization (▶ Fig. 26.4). Magnetic resonance imaging and angiography (MRI/A) provide abundant information about associated structural changes to the brain parenchyma including atrophy, infarcts, calcifications, ventricular size, and the angioarchitecture of the malformation (▶ Fig. 26.5 and ▶ Fig. 26.6). In older children and adults computed

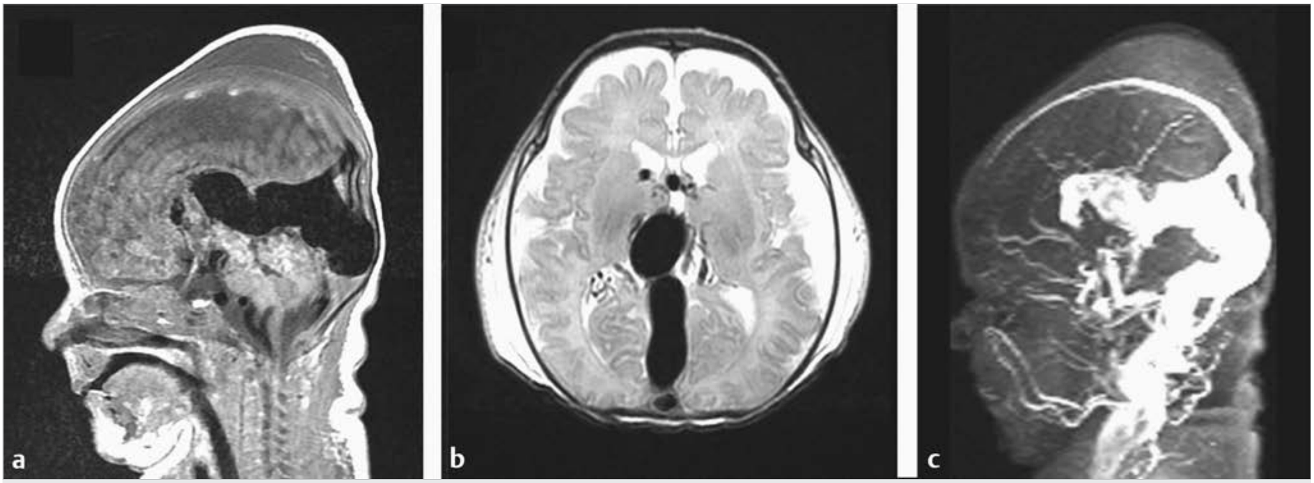

Fig. 26.6 Magnetic resonance imaging of a vein of Galen malformation. **(a)** Sagittal T1-weighted, gadolinium-enhanced image shows the flow void of the vein of Galen and distal falcine sinus and enhancing flow voids anteriorly and inferiorly that represent feeding arterial supply. **(b)** Axial T2-weighted image shows flow voids of the venous drainage and arterial supply. **(c)** Magnetic resonance angiography showing the dilated venous drainage system.

tomography (CT) may be employed as a screening tool and to rapidly diagnose associated subarachnoid or parenchymal hemorrhage. CT angiography (CTA) provides greater spatial resolution and shorter acquisition time than MRA with the downside of additional radiation exposure.[1] In patients with depressed mental status without acute hydrocephalus or hemorrhage, electroencephalography serves as a useful diagnostic tool to rule out status epilepticus and for localization of seizure foci. Catheter angiography remains the gold standard for obtaining detailed anatomic characterization of VGAMs and is typically used once a decision has been made to proceed with endovascular intervention.

26.2.3 Indications for Treatment

Decisions regarding if and when to intervene on a patient harboring a VGAM can be complex. With modern endovascular techniques these lesions no longer carry a uniformly grim prognosis. Cure or palliation is attainable in nearly 80% of patients.[2,3,11] Conversely, in utero evidence of cardiac failure, brain damage, and multiorgan failure at birth are predictive of extremely poor therapeutic outcomes.[3] Poor clinical exam, severe heart failure, extensive intracranial calcifications, and microcephaly portend a poor prognosis and may suggest not treating the VGAM. Infants, children, and adults generally present with less fulminant manifestations thus allowing the clinician more time to treat the lesion on an elective basis. Developmental delay, epilepsy, dementia, congestive heart failure, macrocephaly, headache, intracranial hemorrhage, and hydrocephalus are common indications to treat VGAMs outside the neonatal period.

Lasjaunias et al devised a scoring system to guide clinicians as they contemplate treatment for neonates diagnosed with VGAMs.[3] The Bicêtre neonatal evaluation score translates cardiac, cerebral, respiratory, hepatic, and renal function parameters to a score ranging from 0 to 21. Scores of less than 8 favor a decision not to treat due to extremely poor prognosis, scores of 8 to 12 merit emergent endovascular treatment, and scores greater than 12 suggest medical management and optimization with definitive treatment deferred until the patient is at least 5 months old. In patients with favorable scores, any sign of failure to thrive should prompt reevaluation and possible early embolization.

26.3 Therapeutic Approaches

Most VGAM are treated endovascularly. In rare cases, open ligation of arterial feeders may be performed, typically in the setting of inability to adequately decrease fistulous flow endovascularly. Stereotactic radiosurgery provides a noninvasive therapeutic approach for stable patients wherein nonurgent intervention is appropriate (▶ Fig. 26.7).[12] In some patients, it may be necessary to employ a multimodal staged approach that gradually decreases drainage and allows for stabilization of systemic dysfunction prior to definitive treatment. For embolization in particular, some practitioners prefer staging, reasoning that gradual occlusion minimizes the risk of hemorrhagic or ischemic complications.

26.3.1 Medical Management

Medical management alone does not provide definitive treatment of VGAMs. When possible, medical optimization of the patients prior to undertaking endovascular or surgical intervention is desirable. Neonates presenting with heart failure pose the greatest challenge in this arena (▶ Fig. 26.3). High output cardiac failure from VGAMs affect many organ systems, each of which deserves special attention. Patients often have concomitant renal and hepatic dysfunction due to impaired perfusion. Myocardial ischemia may be present and increased cardiac output may delay closure of the ductus arteriosus thus worsening hypoxemia from volume overload.[1] Congestive failure can lead to pulmonary hypertension, further exacerbating heart failure.

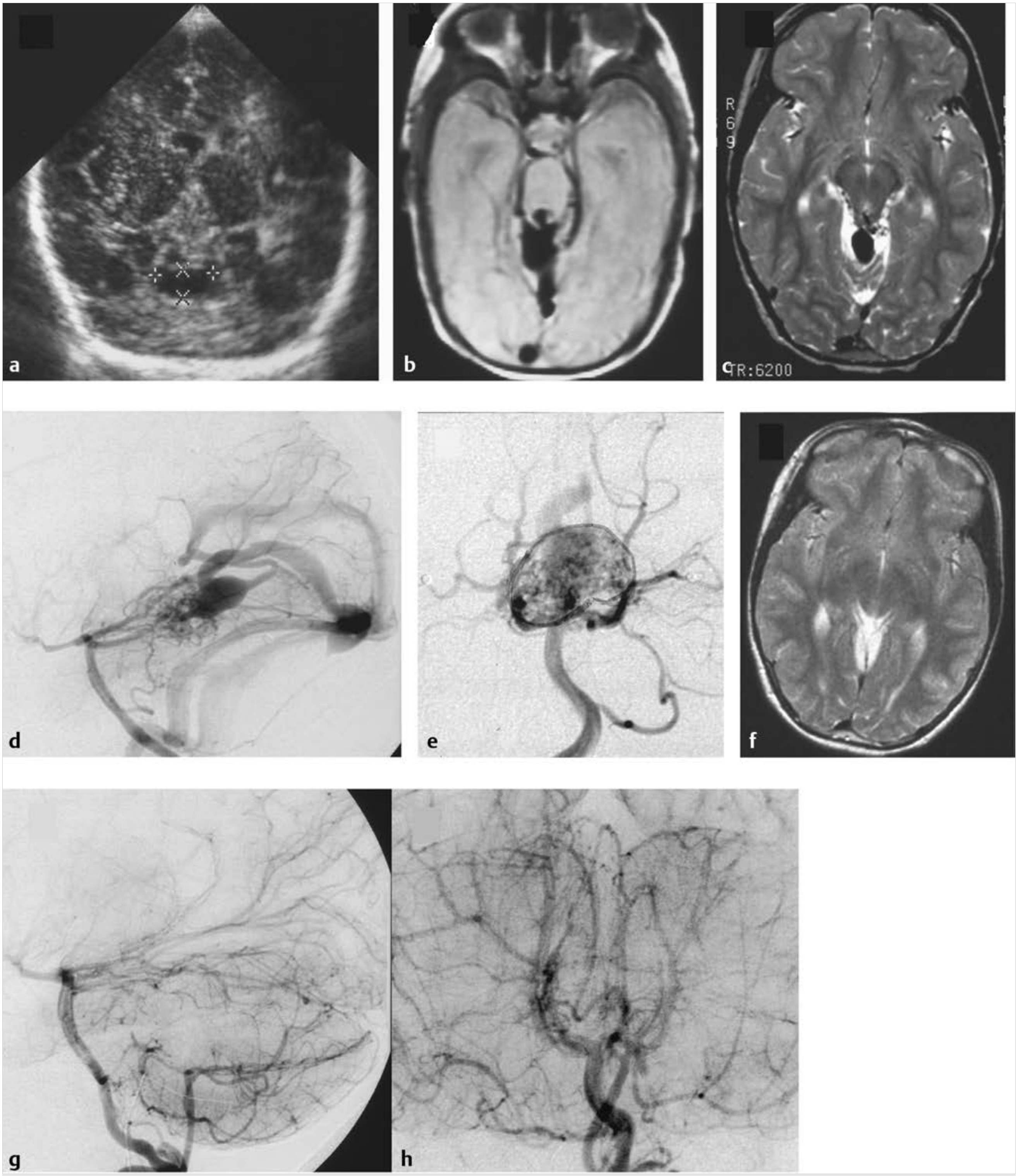

Fig. 26.7 Stereotactic radiosurgery of a vein of Galen malformation. **(a)** Ultrasound at birth showing the flow void of the malformation. **(b)** Magnetic resonance imaging (MRI) at birth shows the malformation. **(c)** The malformation remains unchanged at 3 years of age on MRI. **(d)** Lateral and **(e)** anteroposterior vertebral catheter angiography showing the malformation that was treated with linear accelerator-based stereotactic radiosurgery. **(f)** Three years later MRI and **(g)** lateral and **(h)** anteroposterior vertebral catheter angiography showing obliteration of the malformation. The patient remained neurologically intact.

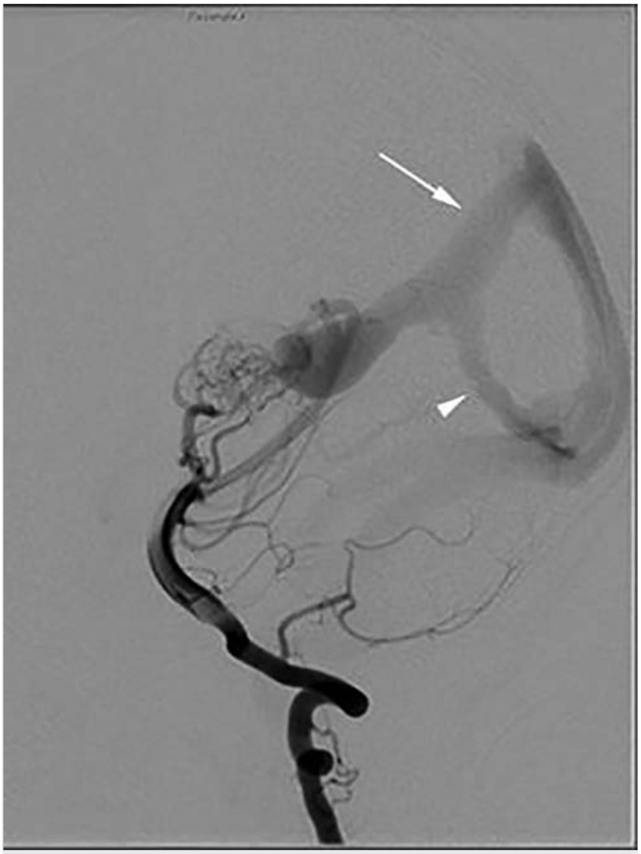

Fig. 26.8 Vertebral arteriography demonstrates the presence of a choroidal vein of Galen aneurysmal malformation draining to a superiorly projecting persistent falcine sinus (*arrow*). A comparatively diminutive straight sinus directed more inferiorly within the tentorium is also present (*arrowhead*).

In prenatally diagnosed patients with severe heart failure on echocardiography, maternal digoxin may be administered until delivery. Upon delivery the patient is managed with inotropes and diuretics to decrease flow and relieve volume overload. By using these measures, many patients can be stabilized to allow delayed treatment at several months of age when they are grown and better able to tolerate intervention. In cases where patients are unable to be weaned from mechanical ventilation or are unresponsive to medications, palliative subtotal endovascular occlusion of the fistula may be indicated.[1,5,9]

26.3.2 Endovascular Approaches

The morbidity and mortality associated with treating VGAMs have decreased with modern endovascular techniques. Staged intervention employing a stepwise reduction in flow to allow gradual hemodynamic normalization is desirable. Such an approach may minimize the risk of normal perfusion breakthrough hemorrhage and deep venous thrombosis during the course of therapy.[1,13] VGAMs may be approached via transarterial, transvenous, or combined routes dictated by the angioarchitecture of the VGAM and by surgeon preference. Many groups favor pure transarterial approaches when possible due to reported higher rates of hemorrhagic complications and the greater average number of embolization sessions with transvenous approaches.[10]

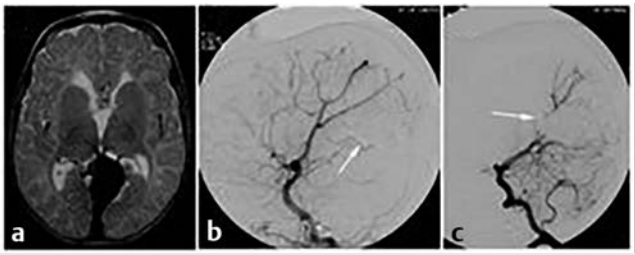

Fig. 26.9 Transvenous embolization with thrombogenic platinum coils (*arrow*) as well as transarterial embolization with n-butyl cyanoacrylate were used to completely occlude this vein of Galen aneurysmal malformation after three staged interventions **(a–c).**

To accomplish safe transvenous VGAM embolization, it is important to be sure that the venous collector is not responsible for substantial normal deep venous drainage. Making this determination can sometimes be difficult; however, when assessing venous drainage patterns it should be kept in mind that that true VGAMs typically empty into a persistent falcine sinus. The straight sinus is often not visible due to stenosis or congenital absence (▶ Fig. 26.8).

The site of vascular access is often dictated by the age and size of the patient. While transfemoral puncture is typically preferred, alternative routes exist. In neonates, the umbilical vein or artery may be accessed if necessary.[13,14] Direct carotid artery, jugular vein, and transtorcular venous puncture may rarely be required.

Following induction of general anesthesia, endotracheal intubation, and systemic heparinization, we typically access the femoral vessels using a modified Seldinger technique with a micropuncture needle. In neonates and infants, a 4 French femoral sheath is usually adequate. Arteriography delineates feeders that may be candidates for embolization as well as assists in the mapping of venous drainage should transvenous occlusion be considered. In some circumstances a transvenous microcatheter may be advanced through the fistula to the arterial side, allowing retrograde embolization to be performed.

Detachable microcoils and liquid agents are the most commonly used materials to accomplish VGAM embolization and thrombosis. Liquid embolic agents including, n-butyl cyanoacrylate (nBCA, Trufill, Codman Neurovascular, NJ) and ethylene vinyl alcohol copolymer (Onyx, Micro Therapeutics, CA) are commonly used transarterially. Liquid agents may be somewhat more advantageous for transarterial embolization in that there exists the potential for more extensive feeder penetration with a lower risk of vessel rupture.[5] In particular, Onyx, which has emerged as a safe and effective alternative to nBCA, has been used to occlude large portions of fistulas through single arterial injections. In cases of high flow lesions, liquid embolic agents can be challenging to use. Embolization of these agents to the pulmonary circulation can be fatal. Its utility through purely transvenous approaches has been documented, but requires further study.[15,16] Appropriately sized thrombogenic platinum coils are ideal for transvenous embolization of the dilated venous collector in part because they have a lower risk of pulmonary embolization than liquid agents (▶ Fig. 26.9 **and** ▶ Fig. 26.10).

Following therapy, the patient should be monitored in a neurological intensive care unit for at least 24 hours with strict blood pressure control and special attention paid to the possible

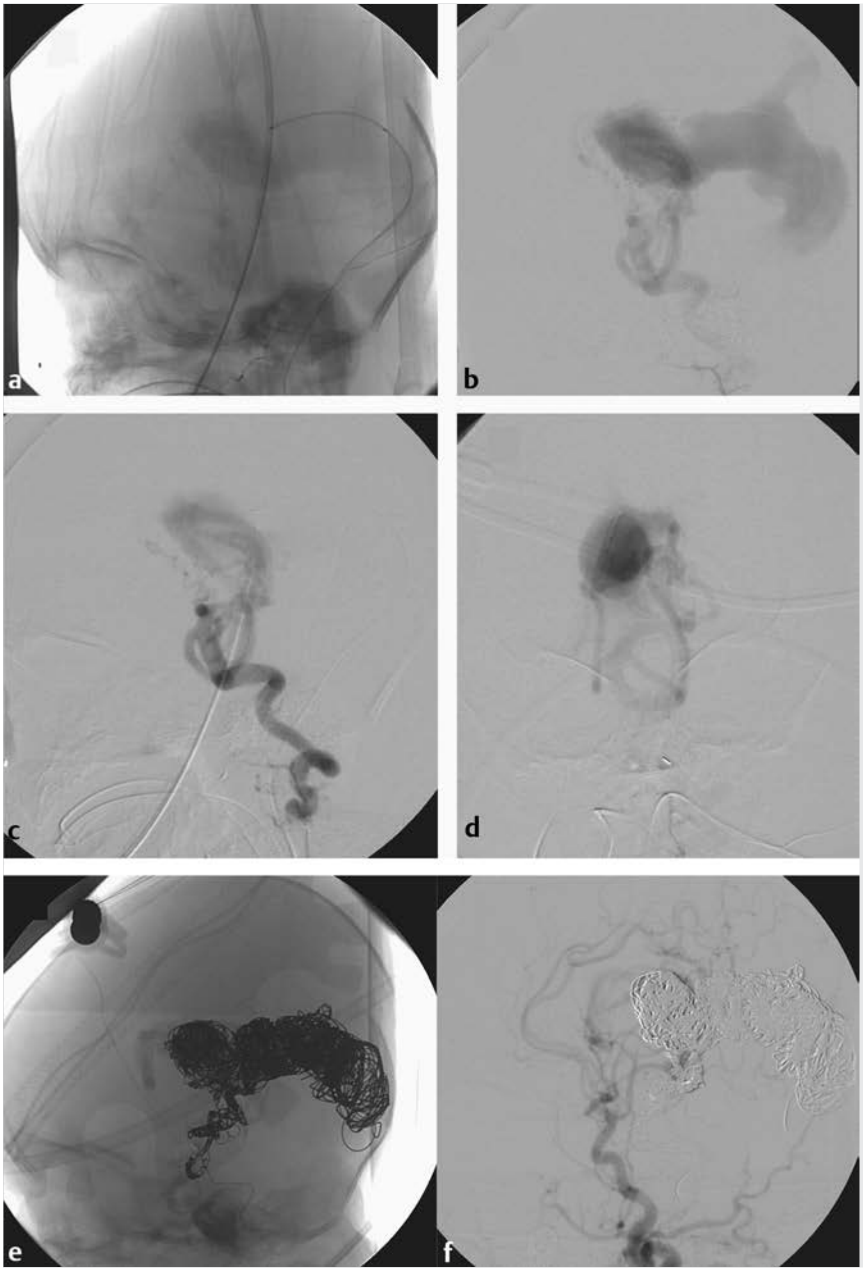

Fig. 26.10 (a, b) Lateral venous phase and **(c, d)** anteroposterior arterial phase vertebral artery angiography demonstrating vein of Galen malformation. **(e)** Unsubtracted lateral skull radiograph after endovascular embolization of a vein of Galen malformation with platinum coils. **(f)** The coil mass is visible, and lateral vertebral angiography shows obliteration of the fistula.

development of consumptive coagulopathy, acid-base disturbances, and renal/cardiac/pulmonary dysfunction.

26.3.3 Surgery

Open surgical treatment of VGAMs is rarely employed but may be considered in the setting of failed or incomplete endovascular occlusion with progressive symptomatology. Open surgical ligation is generally an adjunct to endovascular occlusion or entertained only when endovascular techniques have been unsuccessful. VGAMs may be approached through a posterior interhemispheric approach after parasagittal parieto-occipital craniotomy (▶ Fig. 26.11). A craniotomy that crosses the midline, exposing the superior sagittal sinus allows for adequate falcine exposure after flapping the dura contralaterally. The aneurysm sac is exposed through the callosal, quadrigeminal, and ambient cisterns and arterial feeders are identified at their entrance into the venous sac and sequentially ligated and divided. Palliative, subtotal occlusion of the largest arterial feeders may be all that is required to attain symptomatic relief or enable definitive endovascular treatment. Care should be taken to watch for the development of acute volume overload during progressive occlusion of large fistulas, especially in neonates and small children.

Adjunctive surgical therapy to address hydrocephalus or parenchymal hemorrhage is used when appropriate. It should be noted that cerebrospinal fluid diversion has been suggested to increase the risk of hemorrhage and exacerbate neurological symptoms in untreated malformations and thus should not be undertaken as a means for delaying endovascular therapy.[3,17] Hydrocephalus in this population typically resolves with direct treatment of the malformation.

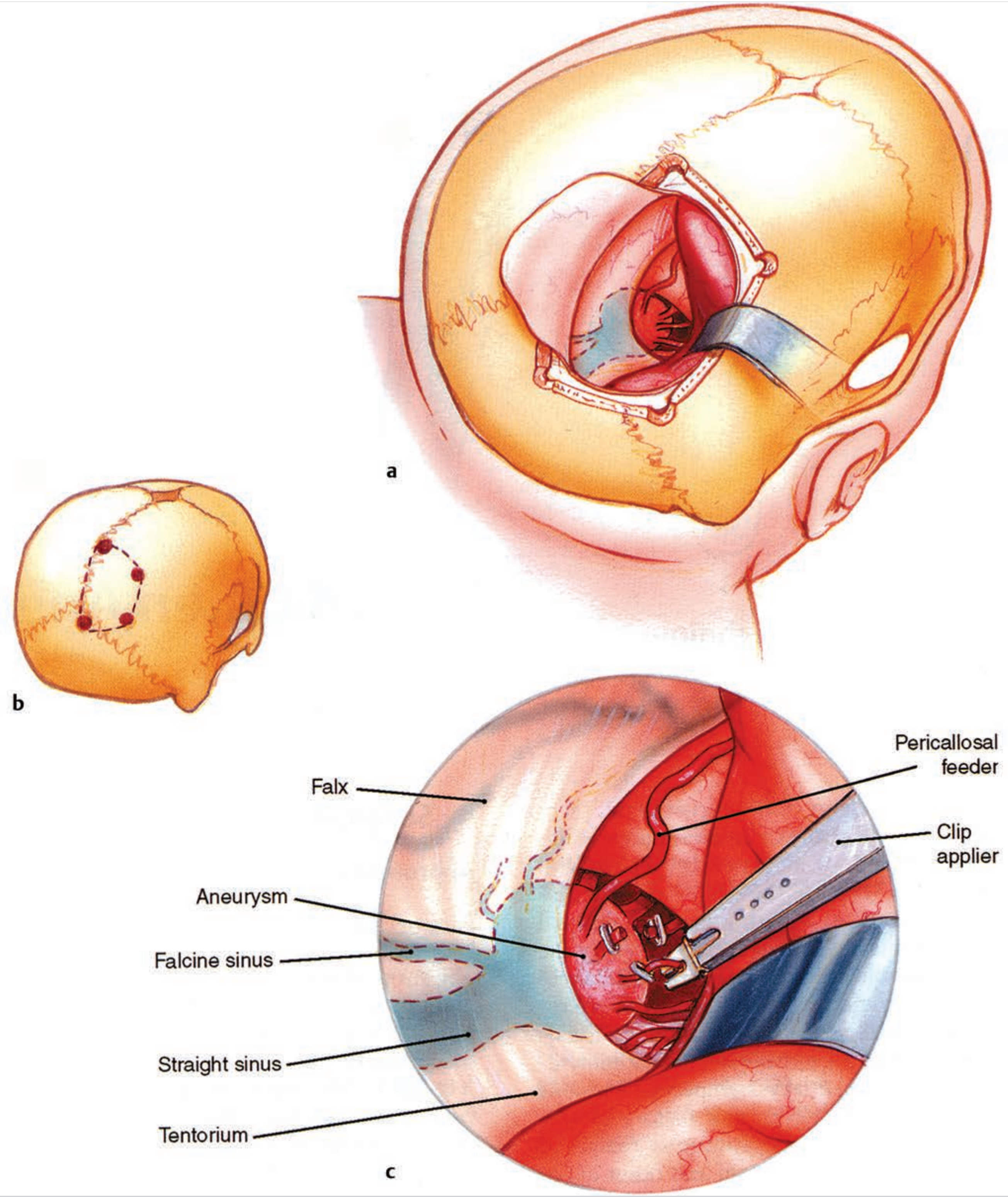
a
b
Falx
Pericallosal feeder
Clip applier
Aneurysm
Falcine sinus
Straight sinus
Tentorium
c

Fig. 26.11 (a–c) Positioning and general technique for posterior interhemispheric approach to a vein of Galen malformation. (Reproduced with permission from Mickle JP. Vein of Galen aneurysms. In: Sekhar LN, Fessler RG, eds. Atlas of Neurosurgical Techniques: Brain. New York: Thieme;2006:316.)

26.3.4 Radiosurgery

There have been few reported cases in the literature of radiosurgery used alone as the primary treatment for VGAMs (▶ Fig. 26.7). It has been suggested as an adjunct to embolization in clinically stable patients, a requirement given the long latency from treatment to observable effect. Radiation doses ranging from 17 to 25 Gy (50% isodose) have shown some success in complex cases resistant to endovascular occlusion. While requiring further study, radiosurgery may be entertained for situations in which a VGAM is unable to be completely occluded endovascularly but it would seldom be indicated as the primary definitive therapeutic modality.[12,18]

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27 Posterior Fossa Arteriovenous Malformations

Jonathan A. White and Babu G. Welch

Abstract

Posterior fossa arteriovenous malformations (AVMs) comprise about 12% of all brain AVMs. About 70% present with hemorrhage into the brain parenchyma, the subarachnoid space, and/or the ventricular system. Current treatment modalities include surgical resection, radiosurgery, and endovascular embolization. Asymptomatic and unruptured AVM can be managed electively with assessment of the risk of the natural history compared to the risk:benefit of treatment. Ruptured posterior fossa AVMs may be associated with acute hydrocephalus and require ventricular drainage and if there is a large cerebellar hematoma, posterior fossa craniotomy, evacuation of the hematoma, and possibly resection of the AVM, depending on the complexity of the AVM. The principles of AVM surgery include adequate exposure, isolation, and control of surface feeding arteries with preservation of venous drainage and arteries en passage followed by circumferential progressively deeper dissection around the AVM, coagulating arterial feeders, and finally transection of venous drainage and completion of removal.

Keywords: cerebral arteriovenous malformation, intracranial hemorrhage

27.1 Patient Selection

About 70% of posterior fossa arteriovenous malformations (AVMs) present with hemorrhage. Headache and incidental detection make up most of the remaining 30% of presentations, and seizures directly caused by posterior fossa lesions are rare. The hemorrhage is most often parenchymal but can also be subarachnoid or intraventricular. The presence of nonparenchymal blood should raise suspicion that the hemorrhage may not be due to the AVM itself but rather to an associated vascular lesion such as an aneurysm on a feeding artery. This occurs in 10% of posterior fossa AVMs.

In patients presenting with hemorrhage the first study obtained is usually a computed tomographic (CT) scan. Cerebellar parenchymal hemorrhage, intraventricular hemorrhage, or subarachnoid blood may be seen. Much less commonly blood is seen within the brainstem itself. A CT angiogram can be obtained at the same time as the initial CT scan once hemorrhage is diagnosed, assuming there are no contraindications to administration of radiographic contrast. Serpiginous vasculature in proximity to the hemorrhage suggests the etiology of the hemorrhage is an AVM. Magnetic resonance imaging (MRI) is useful to better define the anatomy of the hemorrhage and any underlying cause. A catheter angiogram including external carotid injections should be done acutely in case urgent surgery is necessary. Radiographic studies should be scrutinized to determine the location and size of the lesion, the vascular supply, and drainage as well as the presence of any associated vascular abnormalities. Thorough knowledge of the venous drainage pattern is helpful in the planning of surgical and adjunctive therapy.

27.2 Indications and Contraindications for Surgery

The most important factors that determine whether to treat an AVM are the grade of the AVM and whether or not it has ever hemorrhaged (▶ Table 27.1, ▶ Fig. 27.1, ▶ Fig. 27.2, ▶ Fig. 27.3).[1,2] The goal of treatment is to completely obliterate the AVM so that there is no arteriovenous shunting. Treatment is more indicated if there is a history of hemorrhage and low-grade lesions, whereas as the grade increases or if there is no history of hemorrhage then the risk of the natural history which is the risk of hemorrhage begins to be outweighed by the risk of treatment.

Table 27.1 The Spetzler–Martin and Spetzler–Ponce grading systems for brain arteriovenous malformations.[1,2] Spetzler–Martin grade is calculated by summing the points (grade is the sum of the allocated points)

Spetzler–Martin grading system points allocation	
AVM-related features	**Points**
Size	
Small (< 3 cm)	1
Medium (3–6 cm)	2
Large (> 6 cm)	3
Situation	
Non-eloquent area	0
Eloquent area[a]	1
Venous Drainage	
Superficial only	0
Deep draining vein(s)	1

[a] Eloquent areas: sensorimotor, visual, and language cortices; diencephalon; internal capsule, brainstem, cerebellar peduncles, and deep cerebellar nuclei

Spetzler–Ponce classes		
Class	Spetzler–Martin grade	General management
A	1, 2	Usually single modality treatment (surgical resection, stereotactic radiosurgery, complete embolization)
B	3	Multimodality treatment
C	4, 5	No treatment (exceptions include recurrent hemorrhage, neurological deficits steal-related symptoms and associated aneurysms)

If the AVM has ruptured, surgical treatment of the lesion is best delayed until some of the hemorrhage has resolved and the acute cerebral edema has lessened. Waiting 6 to 8 weeks can dramatically decrease the difficulty of surgery and minimizes the chances of injury to the surrounding cerebellum. The rate of recurrent hemorrhage from the AVM may be slightly increased during this period compared with the risk of bleeding from an unruptured AVM or one that has not bled within 6 months, but in general AVMs do not have the same morbid natural history as an untreated aneurysm. Thus the benefits of delayed surgery are thought to outweigh the risks of rebleeding. Emergency surgery includes insertion of an external ventricular drain (EVD) in patients with neurological compromise due to acute hydrocephalus. Cerebellar hematomas greater than 3 cm in maximum diameter or more than about 10 mL volume are usually associated with acute hydrocephalus, brainstem compression, and neurological deterioration and need to be evacuated emergently.[3] The AVM can be resected at the same time if it has been adequately defined by preoperative angiographic imaging and is judged to be a simple surgical lesion (▶ Fig. 27.4**, Video 27.1**). If an associated vascular lesion such as a feeding artery aneurysm is the cause of the rupture it can be treated at the time of craniotomy if it is near enough to the AVM to reach through the same exposure, otherwise it can generally be treated prior to treatment of the AVM (▶ Fig. 27.1).

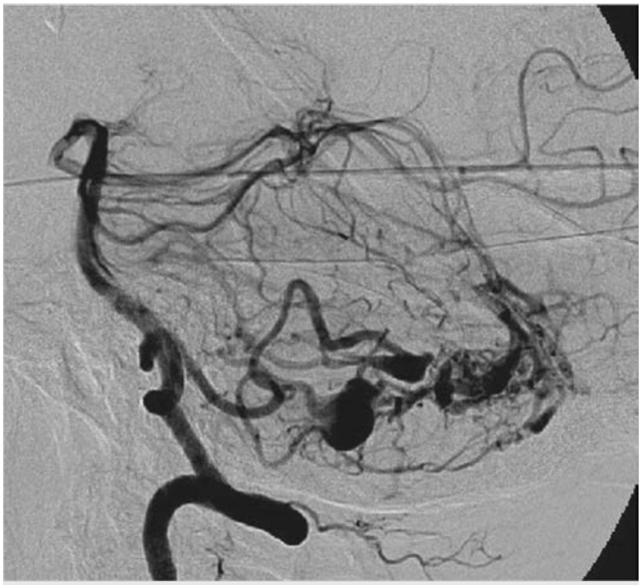

Fig. 27.1 An aneurysm on the distal part of the posterior inferior cerebellar artery that is a feeding artery to a cerebellar hemispheric arteriovenous malformation.

Stereotactic radiosurgery is another option for the treatment of posterior fossa AVM. Deep-seated lesions, particularly true brainstem parenchymal lesions, which often result in significant new neurological deficits when treated surgically, are the lesions most often selected. Partial embolization of the AVM using metal coils, polyvinyl alcohol particles, n-butyl cyanoacrylate, or Onyx (Medtronic, Irvine, CA) is another treatment strategy occasionally employed. Embolization is best considered a surgical adjunct to treat aneurysms that can be the source of hemorrhage but occur on more proximal portions of the feeding vessels. In these cases endovascular “control” can be indispensible. The results of incomplete embolization have been disappointing because it does not lower the recurrent hemorrhage rate and in fact may result in dramatic recruitment of new feeding vessels, making later surgical resection much more difficult. Embolization is probably best reserved as a preoperative strategy for resection of the AVM. When size and the significant involvement of eloquent areas predict substantial surgical morbidity, combinations of endovascular and radiosurgical therapy with a potential for partial resection of these lesions have been reported. It is not yet clear that this multimodality therapy is effective, and attempts at this should probably be reserved for repeatedly symptomatic lesions.

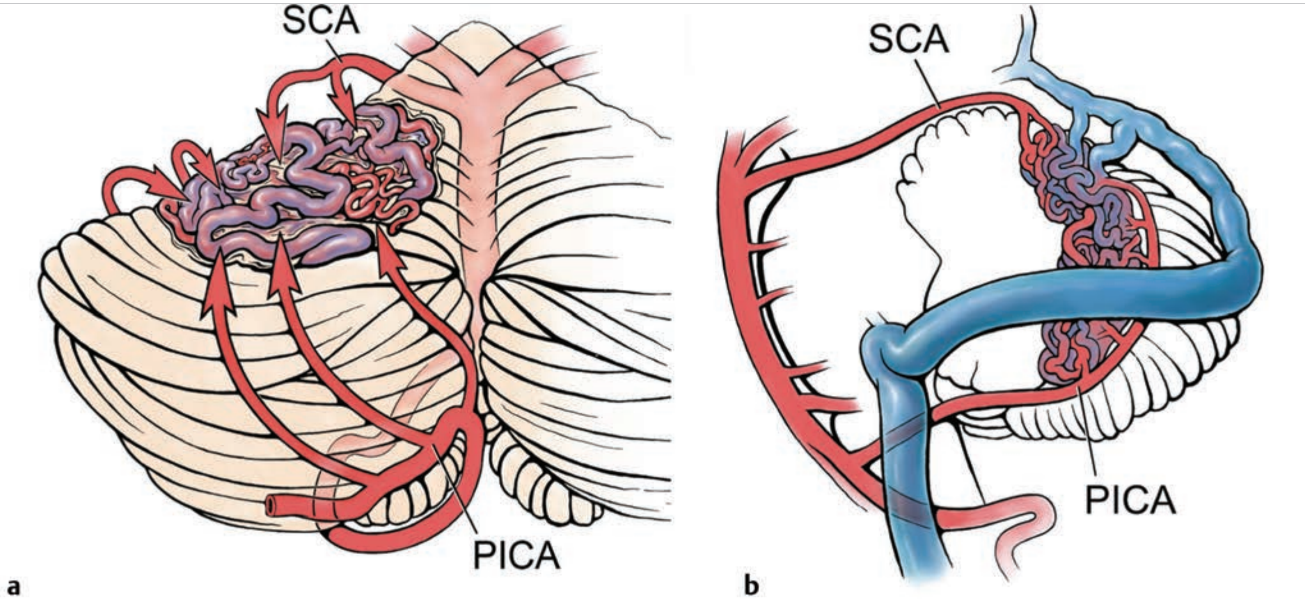

Fig. 27.2 **(a)** A typical arterial feeding to a cerebellar hemispheric arteriovenous malformation (AVM) with supply from the superior cerebellar artery (SCA) and the posterior inferior cerebellar artery (PICA). **(b)** Arterial feeding to an AVM in the cerebellar vermis with supply from the SCA and PICA.

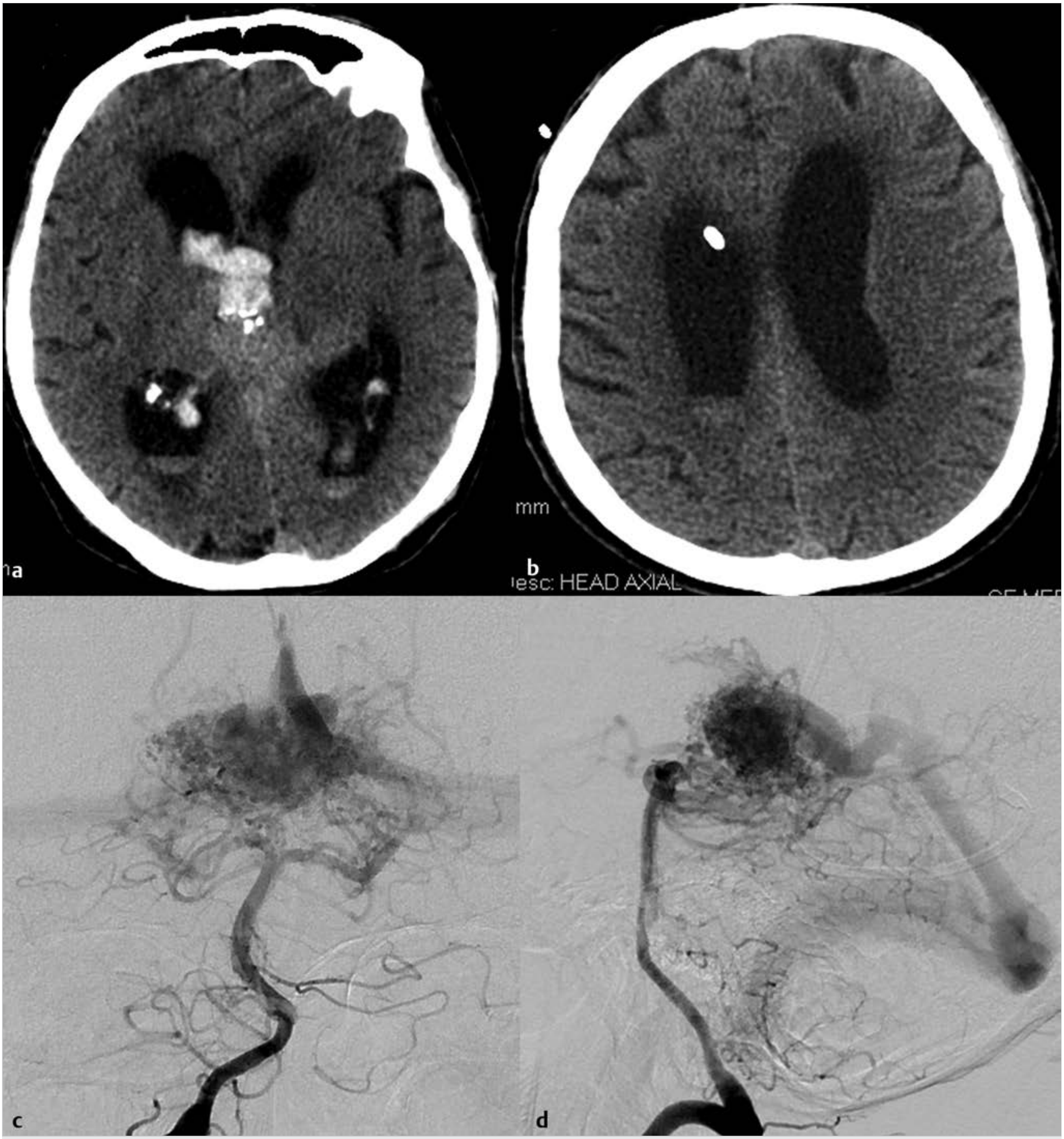

Fig. 27.3 This 76-year-old man presented with a sudden decreased level of consciousness. Computed tomography (CT) scan of the head **(a)** showed an intraventricular hemorrhage, associated hydrocephalus and a thalamic/midbrain lesion suggestive of an arteriovenous malformation (AVM). A CT angiogram was done and a ventricular drain inserted, resulting in neurological improvement **(b)**. Anteroposterior and lateral vertebral cerebral angiography **(c, d)** revealed a high grade (Spetzler–Martin grade 5 or 6), essentially inoperable diencephalic AVM with deep venous drainage. Associated intranidal and distal flow-related microaneurysms were identified, however, they were not amenable for endovascular repair. After removal of the ventricular drain, hydrocephalus and cognitive dysfunction persisted and a programmable ventriculoperitoneal shunt was performed. Follow-up assessments showed very good functional recovery to living at home with minimal assistance with daily living activities due to poor short-term memory.

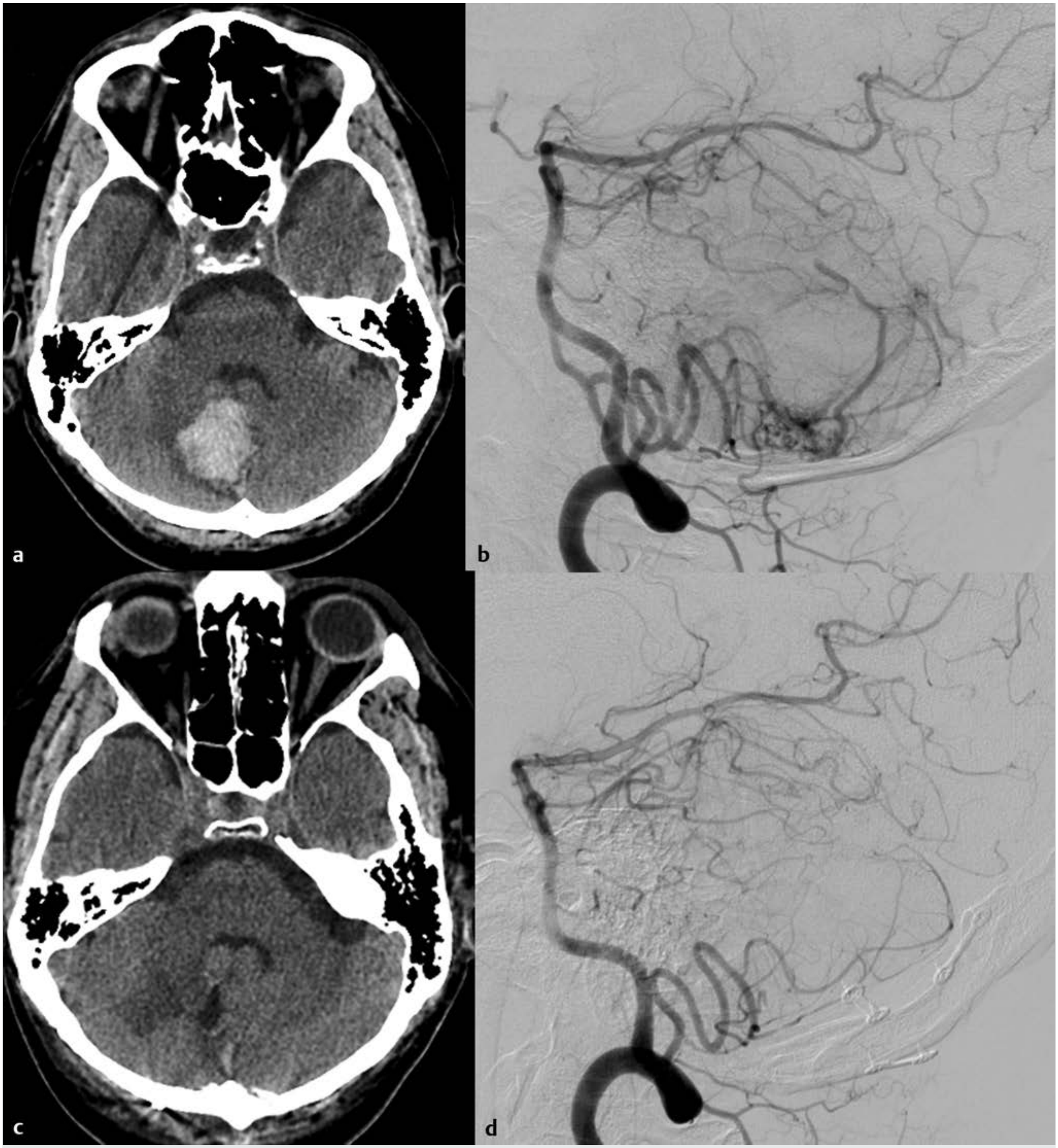

Fig. 27.4 This 58-year-old man presented with a sudden onset of headache, nausea, and vomiting without loss of consciousness. He was on warfarin because of a prior deep vein thrombosis that developed after liver transplantation for hepatitis C, all occurring 6 months ago. Computed tomographic scan of the head showed a right cerebellar hemispheric hemorrhage extending into the vermis **(a)**. Cerebral angiography **(b)** revealed a Spetzler–Martin grade 1 AVM within the right inferior cerebellar hemisphere, fed by right posterior inferior cerebellar artery branches and drained by superficial bilateral cerebellar hemispheric veins and a vermian vein. Because of the hematoma location in the posterior fossa and the arteriovenous malformations (AVM) size, accessibility, and straightforward anatomy, a suboccipital craniotomy with simultaneous AVM excision and hematoma evacuation was performed 48 hours after the hemorrhage when anticoagulation was reversed (**Video 27.1**). The patient's symptoms improved, and follow-up neuroimaging assessments **(c, d)** revealed complete removal of the AVM. He recovered fully and remained off warfarin since it had been 6 months from venous thromboembolism.

27.3 Preoperative Preparation

27.3.1 Classification

Categorizing posterior fossa AVMs by location helps predict the blood flow pattern and is an important part of surgical planning. Posterior fossa AVMs can be categorized by location into cerebellar vermis, cerebellar hemisphere, cerebellar tonsil, superficial pial brainstem, and deep parenchymal brainstem. AVMs of the vermis receive bilateral superior and posterior inferior cerebellar artery supply, whereas hemispheric lesions have a unilateral supply (▶ Fig. 27.2). If a vermian AVM is large enough to reach the roof of the fourth ventricle then arterial supply from the anterior inferior cerebellar artery will be seen. Superior vermian lesions, above the horizontal fissure, receive predominantly superior cerebellar artery feeding, whereas inferior lesions are predominantly fed by the posterior inferior cerebellar artery. Drainage is usually directed through superior vermian veins bridging to the tentorium and through the precentral cerebellar vein.

The dominant supply to a cerebellar hemispheric AVM depends on its location in the hemisphere, with superior lesions receiving more from the superior cerebellar artery and inferior lesions having their predominant supply from the posterior inferior cerebellar artery. AVMs in the cerebellopontine angle involving the middle cerebellar peduncle or the lateral aspect of the ventricular wall may be supplied by the anterior inferior cerebellar artery. Superior hemispheric AVMs tend to drain into the superior petrosal vein. Malformations of the cerebellar tonsils are less common and receive unilateral posterior inferior cerebellar artery supply, although larger lesions can recruit anterior inferior cerebellar arterial supply. Drainage is usually to inferior vermian veins or into the sigmoid sinus (▶ Fig. 27.5).

Brainstem AVMs are typically of two types—pial and true parenchymal. Superficial pial lesions can be resected with reasonable safety, but true parenchymal lesions carry an exceptionally high risk with microsurgical resection. Feeding of superficial pial AVMs is typically from the superior and anterior inferior cerebellar arteries. The venous drainage is usually into the prepontine and petrosal venous systems. Deep parenchymal brainstem lesions receive arterial input directly from vertebrobasilar perforators penetrating the brainstem substance. Differentiating the arterial supply to the AVM from perforators to normal brainstem is difficult surgically, which creates substantial risk of causing permanent neurological deficits with surgical resection. Venous drainage is through periependymal venous channels into the Galenic system (▶ Fig. 27.6).

27.3.2 Preoperative Embolization

Preoperative embolization can be used to reduce or eliminate deep arterial supply to AVM in order to simplify surgery and reduce blood loss. It is less important as a sole treatment. The focus should be on globally reducing flow to the lesion while eliminating vascular pedicles, which are difficult to access during the early stages of surgery. The posterior inferior and superior cerebellar arteries are often large contributors to these malformations, and so their embolization can serve to reduce global flow. Both of these pedicles are generally easily accessible during the early stages of surgery; therefore, proximal embolization, which could risk occlusion of brainstem perforators, is unnecessary and risky. Anterior inferior cerebellar artery feeding can be difficult to expose early in the course of surgery, thus embolization of these vessels is extremely helpful. While performing embolization as distal as possible is an important principle, it is crucial to adhere to this concept when embolizing through the anterior inferior cerebellar artery. Retrograde thrombosis of the vessel can produce profound dysfunction of cranial nerves VII and VIII.

27.3.3 Anesthetic and Preoperative Management

Surgical treatment is best undertaken weeks after hemorrhage. This allows resolution of the posterior fossa edema and greatly

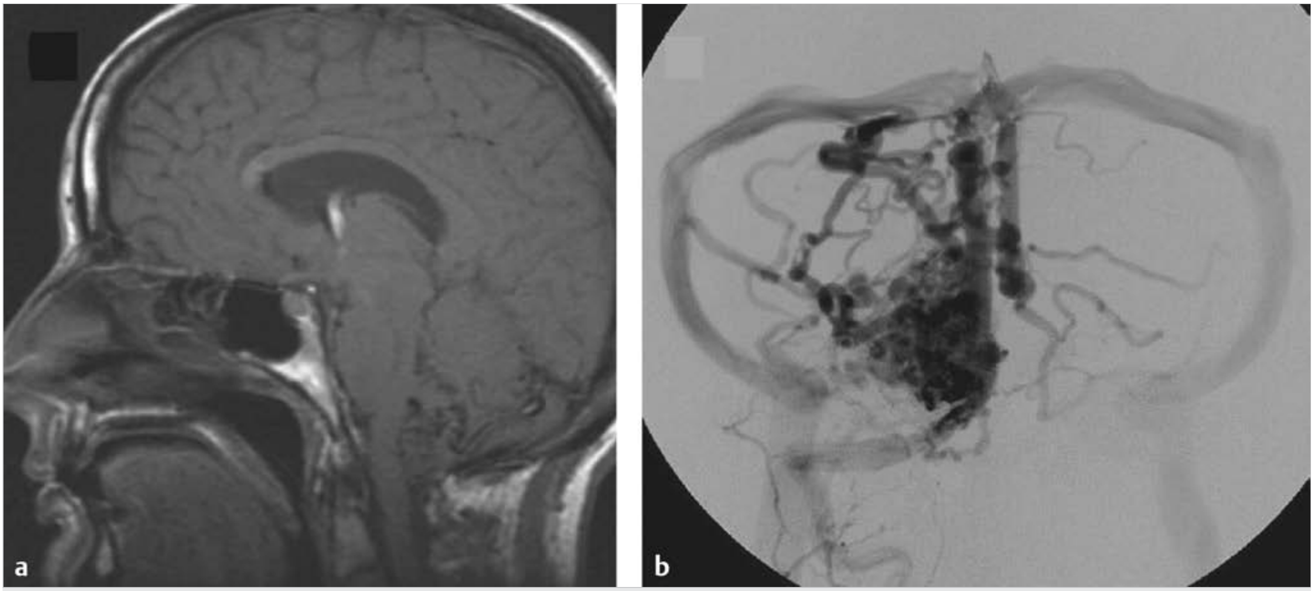

Fig. 27.5 **(a)** Sagittal T1-weighted magnetic resonance imaging of an arteriovenous malformation (AVM) in the cerebellar tonsil. **(b)** Anteroposterior vertebral angiogram of the AVM in the cerebellar tonsil. Arterial supply is principally from the ipsilateral posterior inferior cerebellar artery. Due to its large size there is superior drainage in addition to the more common drainage inferiorly and laterally.

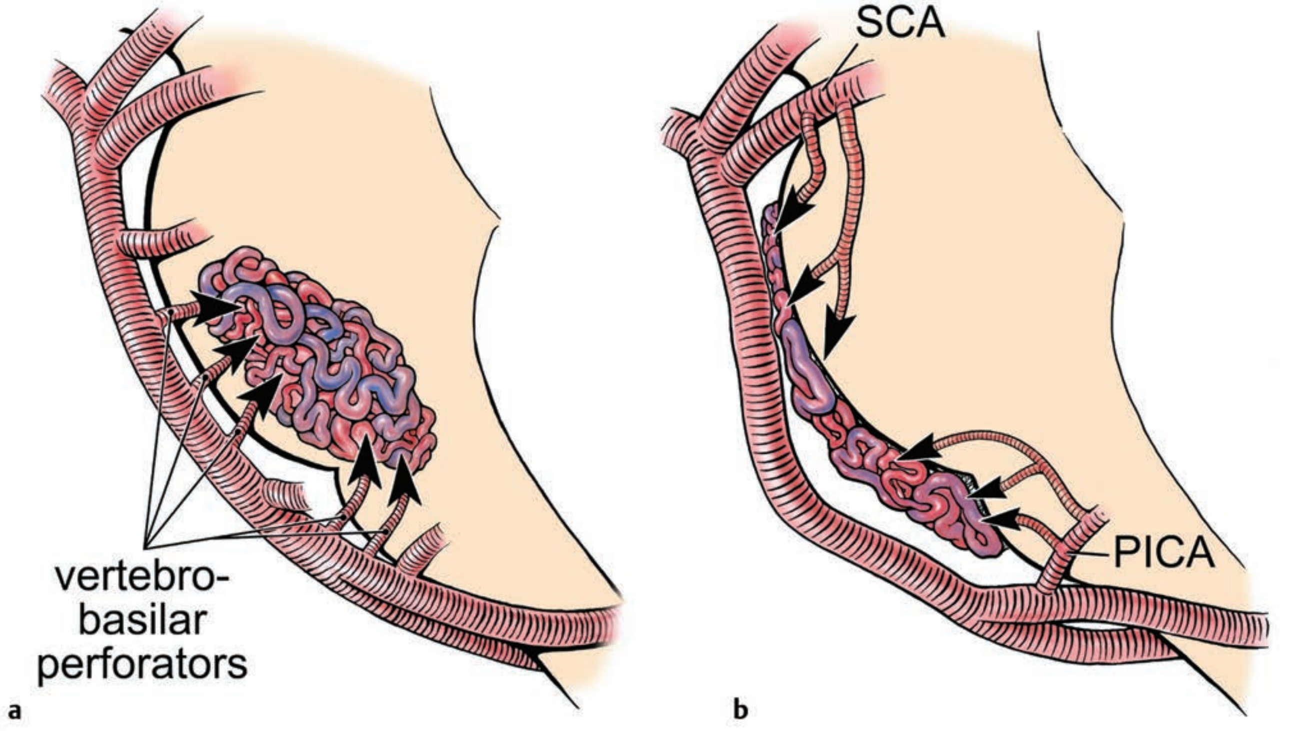

Fig. 27.6 **(a)** A deep parenchymal brainstem arteriovenous malformation (AVM) showing feeding directly from vertebrobasilar perforators. **(b)** Drawing of a superficial pial brainstem AVM. The feeding is superficial and from the superior cerebellar artery and the posterior inferior cerebellar artery.

facilitates exposure. Hydrocephalus may need to be treated in the meantime with either an EVD or permanent cerebrospinal fluid (CSF) diversion. A preoperative EVD should be placed in patients with ventriculomegaly and no CSF shunt. This will help with brain relaxation during surgery and with management of intracranial pressure and wound healing postoperatively. Anticonvulsants are probably unnecessary in the absence of significant supratentorial subarachnoid blood. General anesthesia is required for surgery. After induction, central venous and arterial pressure lines are placed. A second air embolus line may be placed if a semisitting position is to be used. Preoperative antibiotics are given for appropriate gram-positive coverage.

During the procedure systemic blood pressure is kept at low normotensive levels with systolic pressures in the 100 to 120 mm Hg range. Intravenous mannitol is used to facilitate brain relaxation along with opening of the EVD prior to dural opening. Mild hyperventilation to a $PaCO_2$ of 30 mm Hg also helps control brain swelling. A Foley catheter and mechanical prophylaxis against deep vein thrombosis should also be used. For lesions in the brainstem, auditory evoked potential and cranial nerve monitoring may help to reduce postoperative morbidity.

27.4 Operative Procedure

27.4.1 General Principles

The principles of posterior fossa AVM surgery are the same as those for supratentorial AVM surgery and include logical patient positioning, adequate bony exposure, a large dural opening, sharp microdissection, and compulsive hemostasis. Proper physician and patient positioning helps minimize surgeon fatigue and maximize brain relaxation. If extensive work is to be done in the cerebellopontine angle, a lateral position and far lateral exposure optimize the view. Vermian and medial hemispheric lesions are best approached from a midline suboccipital exposure. Bony removal should be extensive enough to allow mobilization of the lesion in multiple directions and to gain access to proximal feeding vessels should the need arise. Similarly, a wide dural opening should be made, with care to inspect the undersurface of the dura during the opening to avoid tearing a draining vein, which may become incorporated into the dura.

After the dura is opened, it is confirmed that the opening is sufficient to remove the lesion safely. A pial incision is then made around the entire lesion demarcating the area of resection. Superficial feeding arteries are divided during this process. The margin in the vascular distribution of those vessels least well embolized should be attacked first. The venous drainage must be preserved until the majority of the arterial input is disconnected. Additional malformation can often be found hidden adjacent to the draining vein. Care should be used not to get too deep on any one margin but to circumferentially deepen the dissection. This is particularly true when approaching ventricular surfaces because the periependymal feeding can be difficult to handle.

27.4.2 Arteriovenous Malformations of the Cerebellar Vermis and Tonsils

AVMs of the vermis and cerebellar tonsils are best handled through a midline suboccipital exposure (▶ Fig. 27.5). Patients are positioned prone on chest rolls with the back elevated. It is helpful to angle the head toward the opposite shoulder. This allows the surgeon better access to the midline without having to

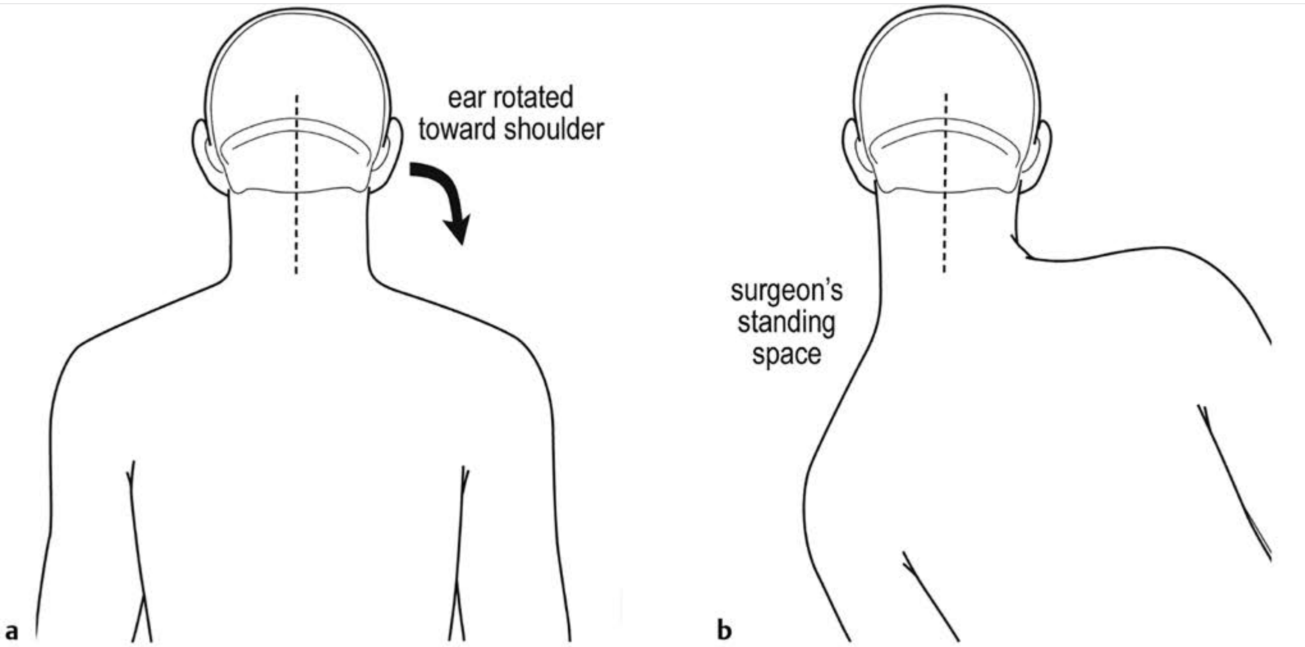

Fig. 27.7 **(a)** Positioning for a prone midline suboccipital exposure. **(b)** Angling the head toward the contralateral ear creates a more comfortable position for the surgeon.

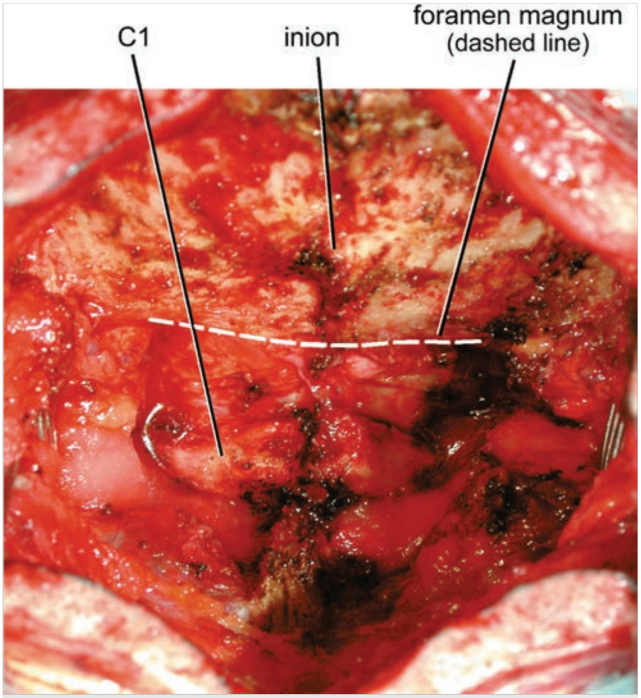

Fig. 27.8 Intraoperative photograph after subperiosteal elevation of the suboccipital musculature. The foramen magnum and posterior arch of the atlas (C1) can be seen.

lean over the patient's back (▶ Fig. 27.7). A midline incision is made from above the inion to the level of the spinous process of the fourth cervical vertebra. Musculature is elevated in a subperiosteal fashion to expose the occipital bone down to the foramen magnum and the posterior arch of the atlas (▶ Fig. 27.8). Bony exposure should include the transverse sinus for superior vermian lesions and the foramen magnum and posterior arch of the atlas for tonsillar lesions. The dura is opened to maximize exposure (▶ Fig. 27.9).

The microscope is then brought in, and the arachnoid of the cisterna magna is opened. This allows CSF to be evacuated and further facilitates brain relaxation. Microdissection allows identification of the posterior inferior cerebellar arteries near the midline; the vessels can be followed to their entry into the lesion and divided at this point. Clip ligation is required because the vessels are usually too large to trust simple bipolar coagulation. Superficial, circumferential pial dissection is then undertaken, eliminating superficial posterior inferior and superior cerebellar feeding arteries (▶ Fig. 27.10). The AVM is usually much softer at this point, with only deep arterial supply remaining from the anterior inferior and superior cerebellar arteries and periependymal feedings. In the final stages the roof of the fourth ventricle is opened to coagulate the remaining feeders. The vein is then divided and the lesion is removed. Pure tonsillar AVMs are smaller, fed only by the ipsilateral posterior inferior cerebellar artery, and can be treated by amputation of the involved tonsil after clip ligation of the posterior inferior cerebellar artery.

27.4.3 Arteriovenous Malformations of the Cerebellar Hemispheres

The optimal patient position for cerebellar hemispheric AVMs depends on the location of the AVM. Lesions near the vermis are best approached via a midline suboccipital approach with the patient in the prone position (▶ Fig. 27.4**, Video 27.1**). Bony exposure to include the transverse sinus or the foramen magnum and the posterior arch of the atlas depends on the position of the AVM relative to the horizontal fissure of the cerebellum. Lesions that are lateral in the hemisphere, particularly those approaching the cerebellopontine angle, are best done in the lateral position with a shepherd's crook incision (▶ Fig. 27.11).

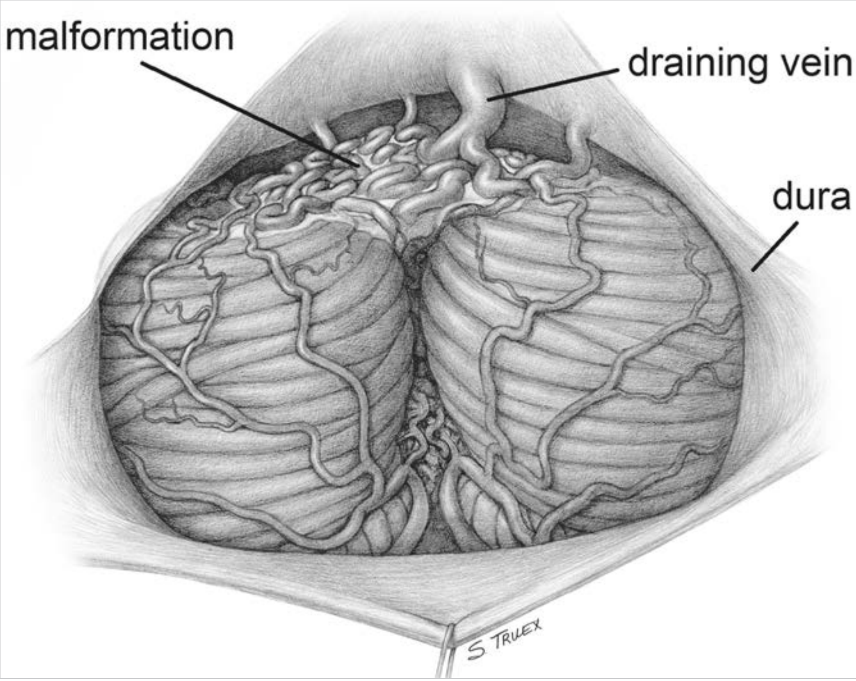
malformation
draining vein
dura
S Truex

Fig. 27.9 The view of a wide dural opening after a midline suboccipital exposure.

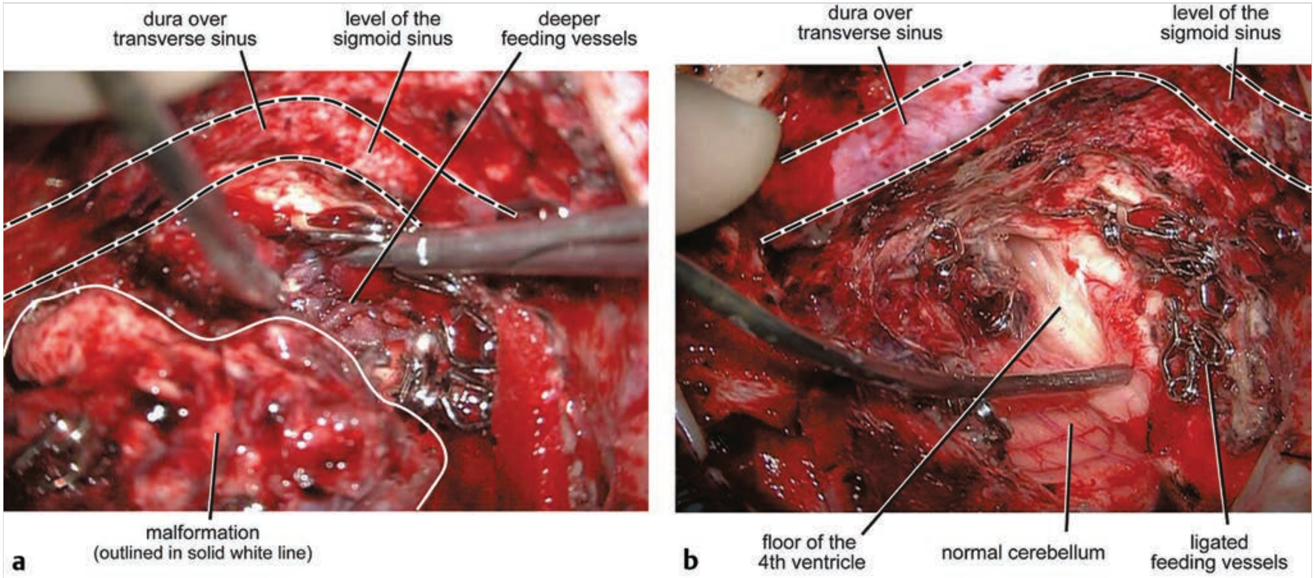
dura over transverse sinus
level of the sigmoid sinus
deeper feeding vessels
malformation (outlined in solid white line)
a
dura over transverse sinus
level of the sigmoid sinus
b
floor of the 4th ventricle
normal cerebellum
ligated feeding vessels

Fig. 27.10 **(a)** Intraoperative photograph during the midportion of resection of an arteriovenous malformation (AVM) of the superior cerebellar vermis. Deep feeding arteries are being divided. **(b)** Intraoperative photograph of the resection cavity after the AVM is removed. The floor of the fourth ventricle is seen.

The superficial muscular layer, including a portion of the sternocleidomastoid, splenius capitus, and semispinalis, is elevated with the skin flap, exposing the deeper musculature, the posterior arch of the atlas, and the extracranial vertebral artery (▶ Fig. 27.12). The superior and inferior oblique muscles are removed from the lateral mass of the atlas, completing the soft tissue dissection. A craniectomy is then performed. The ring of the atlas can be removed with a drill or rongeur (▶ Fig. 27.13). If extensive dissection is to be done in the cerebellopontine angle then the transverse and sigmoid sinuses are exposed. The dura is opened with a flap based along the sigmoid sinus to maximize exposure.

The microdissection again begins with the interruption of the pial surface around the lesion. Superficial superior and posterior inferior cerebellar artery supply is eliminated in this fashion (▶ Fig. 27.14). The deep side of a large lesion will be fed by the anterior inferior cerebellar artery. These vessels are difficult to control early in the dissection and are good candidates for preoperative embolization. Later in the dissection the arterial supply from the anterior inferior cerebellar artery can be identified proximal to the cerebellopontine angle as the branches course over the flocculus or enter the foramen of Luschka to supply the roof of the fourth ventricle. If the preoperative MRI shows that the roof of the fourth ventricle is not involved, the ventricle will not need to be entered to resect the lesion.

27.4.4 Arteriovenous Malformations of the Brainstem

Superficial lesions associated with the pial surface are most common on the anterolateral aspect of the brainstem surface and are fed directly by branches of the anterior inferior and superior cerebellar arteries (▶ Fig. 27.6). Drainage is usually through a lateral pontine vein into the petrosal or Galenic system. These AVMs are often associated with the cerebellopontine angle and so can be approached using the far lateral suboccipital exposure as described earlier for lateral hemispheric lesions.

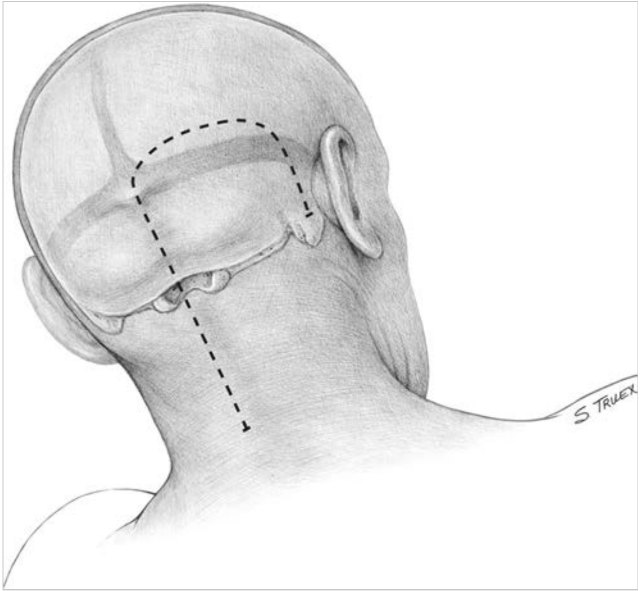

Fig. 27.11 The skin incision for a far lateral, suboccipital exposure.

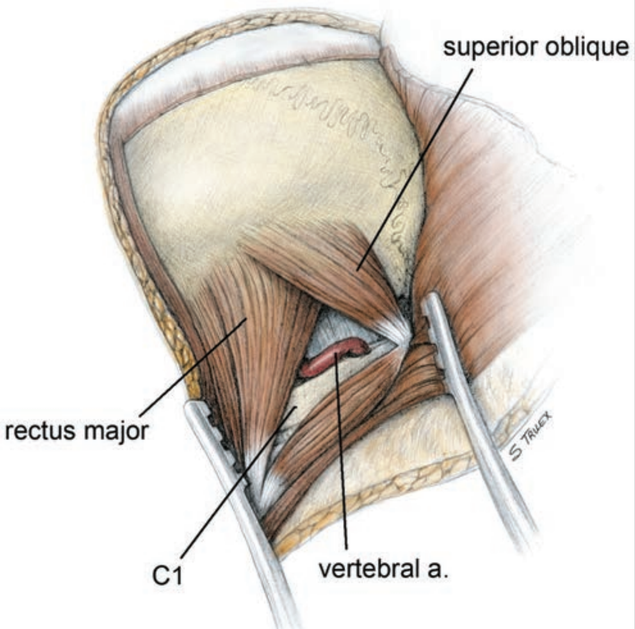
superior oblique
rectus major
C1
vertebral a.
S TRUEX

Fig. 27.12 The view for a lateral, suboccipital exposure after the superficial musculature has been elevated. The extracranial vertebral artery can be seen (C1: posterior arch of the atlas).

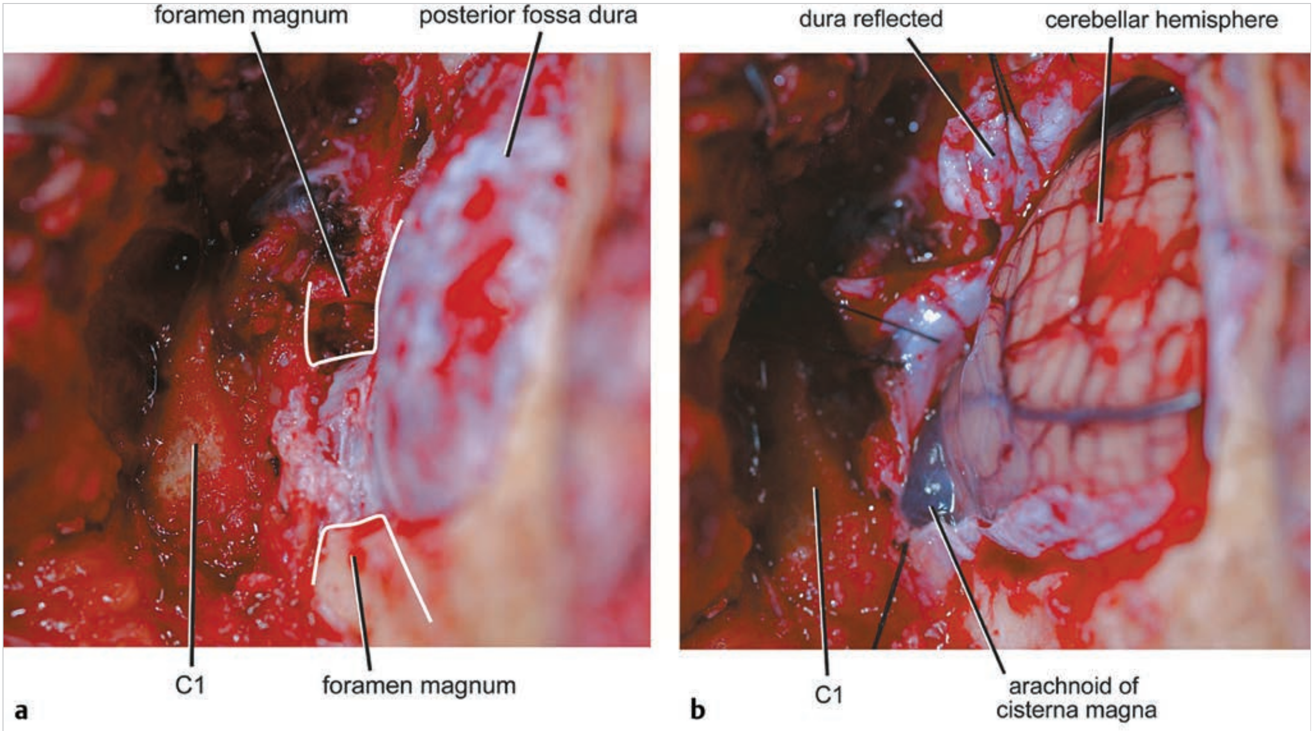
foramen magnum
posterior fossa dura
C1
foramen magnum
a
dura reflected
cerebellar hemisphere
C1
arachnoid of cisterna magna
b

Fig. 27.13 **(a)** Intraoperative view of the dura after craniectomy and removal of a portion of the ring of the atlas (C1) during a lateral suboccipital exposure. **(b)** The dura is opened and the cerebellar hemisphere and cisterna magna can be seen.

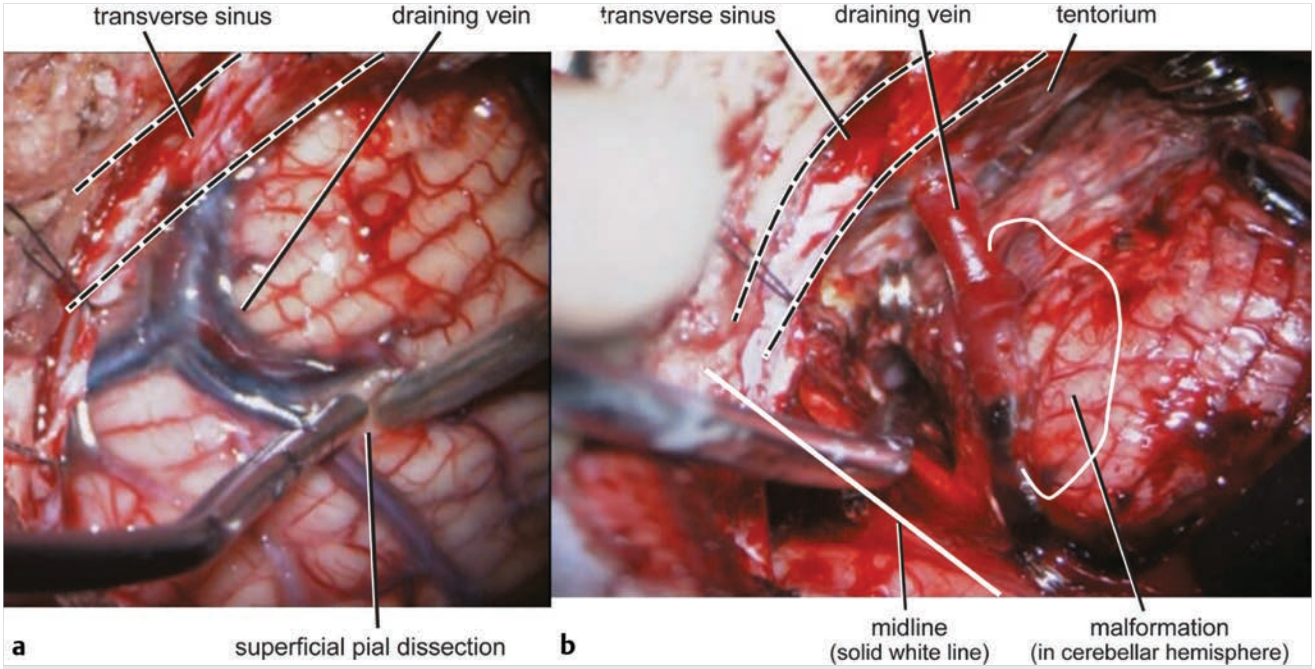

Fig. 27.14 (a) Intraoperative photograph showing the beginning of the superficial dissection of a hemispheric arteriovenous malformation. **(b)** Later in the dissection the venous drainage can be seen entering the tentorium.

The superficial pial surface AVMs tend to have a more favorable surgical outcome than the deep brainstem variety.

Deep brainstem AVMs carry higher morbidity than superficial lesions because their arterial supply comes directly from vertebrobasilar perforators that enter the malformation through normal brainstem tissue. For the most part they are poor surgical candidates unless they are surrounded by a resolving hematoma cavity. The principle of resection is to conserve the maximal amount of surrounding tissue. These lesions are often amenable to radiosurgical therapy.

27.5 Postoperative Management Including Possible Complications

An angiogram obtained while the patient is still anesthetized after surgery allows the option of immediate return to surgery if there is residual AVM identified. This will help minimize the risk of recurrent hemorrhage. If uncontrolled bleeding has occurred during surgery and significant brain swelling exists at the time of closure, the patient can be kept sedated under anesthesia to help control the intracranial pressure.

All patients will be monitored in the intensive care unit. The blood pressure should be controlled tightly with a systolic pressure of 100 to 120 mm Hg for several days to decrease the risk of postoperative hemorrhage. The EVD should be weaned slowly to help ensure that hydrocephalus does not interfere with wound healing. The far lateral suboccipital wound is particularly prone to healing complications and should be monitored closely. New neurological deficits related to resection of functional brain tissue adjacent to the AVM is a common complication and must be explained to the patient and family pre as well as postoperatively. Cerebellar deficits tend to recover better than brainstem and cranial nerve deficits. Hemorrhage into the resection cavity can occur from residual AVM or normal perfusion pressure breakthrough in the adjacent brain and should be treated early by surgical evacuation.

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28 Superficial Cavernous Malformations

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Abstract
Superficial cavernous malformations (SCMs) are brain vascular malformations that occur in the cortex and occasionally in the white matter of the cerebral hemispheres. Studies including about 24,000 brain magnetic resonance imaging (MRI) exams suggest that about 0.5% of the population has a brain CM.[1,2] Roughly 80% are sporadic whereas the remaining 20% are familial and usually associated with one of three genetic loci on chromosomes 7q (*CCM1/KRIT1*), 7p *(CCM2*), and 3q (*CCM3/PDCD10*).[3] The majority of patients with these lesions will be asymptomatic, but they can present with seizures or intracerebral hemorrhage. SCMs are an uncommon cause of intracerebral hemorrhage and therefore infrequently require surgical resection due to their relatively favorable natural history. When SCMs hemorrhage they commonly do so in a cluster over a short time, but they seldom cause severe, permanent neurological disability and even more rarely, mortality. In spite of this relatively favorable natural history, some of these lesions should be considered for surgical resection.

Keywords: cavernous malformation, craniotomy, vascular malformation

28.1 Patient Selection

Typical computed tomographic (CT) characteristics of these lesions include focal or nodular appearing lesions with mild to moderate increase in attenuation without mass effect. Calcification can be noted. Contrast-enhanced CT shows mild to moderate enhancement with heterogeneous mottled appearance and a rim of decreased attenuation consistent with the gliotic tissue surrounding the lesion (▶ Fig. 28.1). On magnetic resonance imaging (MRI), they appear as well-circumscribed popcorn-like lesions (▶ Fig. 28.2). Acute hematoma containing oxyhemoglobin is isointense on T1-weighted images and hypointense on T2-weighted images. Subacute hemorrhage will contain extracellular methemoglobin, which is hyperintense on both Tl- and T2-weighted images. There is usually a heterogeneous core surrounded by a low signal intensity hemosiderin rim on T1-weighted images. The hypointense rim becomes more prominent or "blooms" on T2-weighted and gradient echo sequences. Susceptibility-weighted imaging, which usually uses a gradient echo sequence, is the most sensitive for detection of CMs.[4,5] CMs are not usually associated with mass effect or edema unless there is recent hemorrhage. They are frequently associated with developmental venous anomalies and are generally angiographically occult (▶ Fig. 28.1). The diagnosis can usually be inferred from the MRI appearance and catheter angiography is rarely indicated except when an arteriovenous malformation must be ruled out. CT or MR angiography may help.

28.2 Indications and Contraindications to Surgery

Indications for surgical treatment of superficial cavernous malformation (SCMs) include repeated hemorrhages or medically refractory seizures with electrophysiological localization to the region of the SCM.[4] The location of the lesion (eloquent vs. noneloquent cortex) has to be considered and balanced against the risk of resection. Asymptomatic, incidentally discovered lesions should almost always be left alone. In terms of timing of surgery, it is easiest to resect the lesion by waiting more than 6 weeks after a hemorrhage. The options for management include observation. The role of stereotactic radiosurgery remains controversial and surgical resection is generally favored over radiation.

28.3 Preoperative Preparation

Preoperative imaging should include an MRI with gradient echo sequences/susceptibility-weighted imaging. The main diagnostic

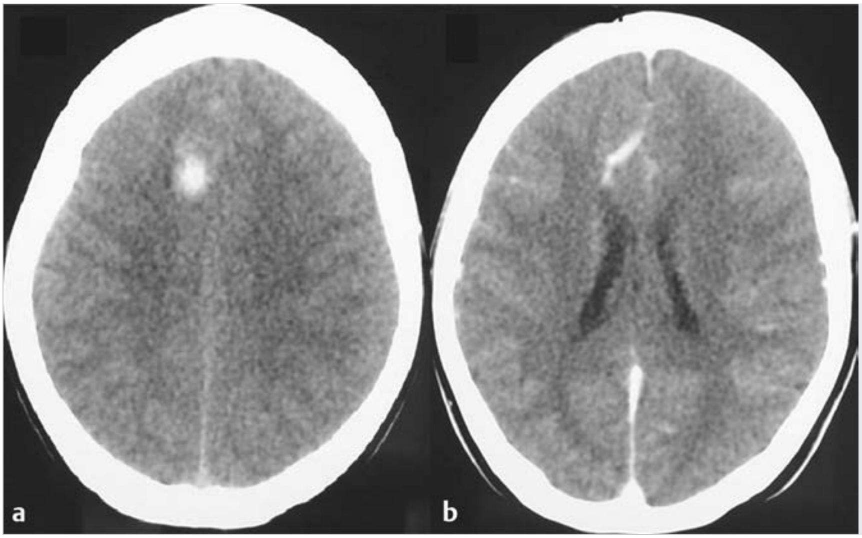

Fig. 28.1 **(a)** Contrast-enhanced computed tomographic scan demonstrating a right frontal hyperdensity associated with **(b)** a vascular structure. These characteristics are consistent with a superficial cavernous malformation and associated developmental venous anomaly.

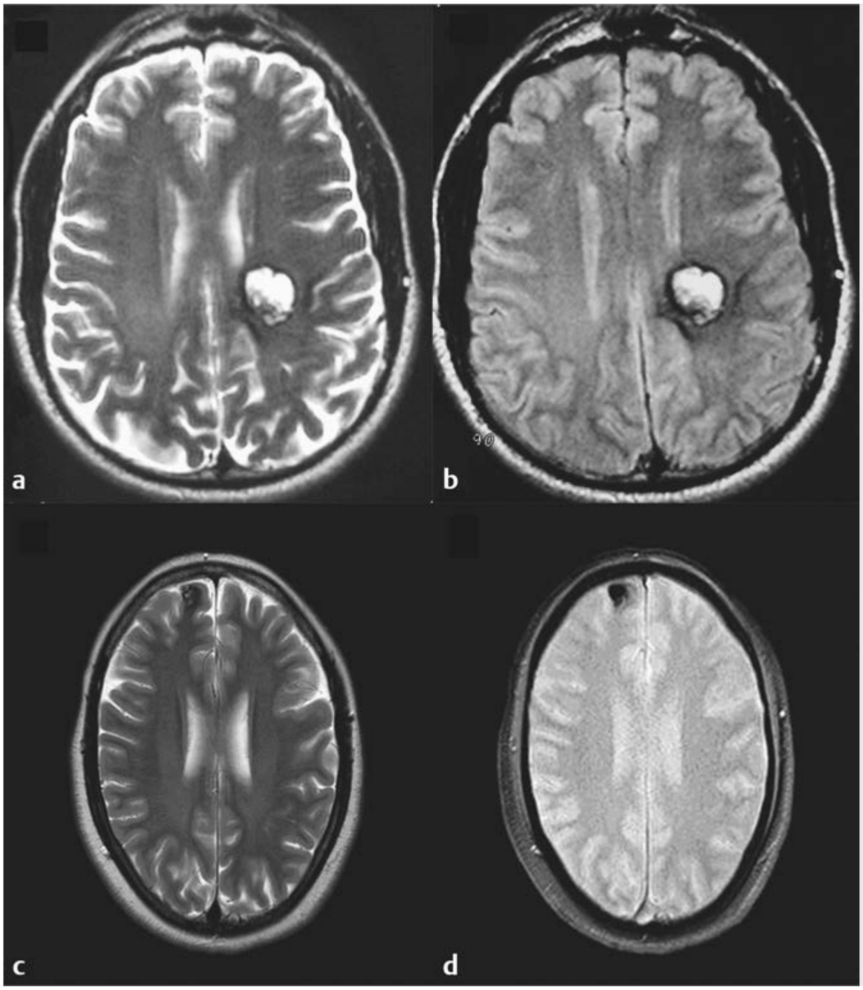

Fig. 28.2 **(a)** Axial T2, **(b)** fluid attenuated inversion recovery images of left parietal cavernous malformation. **(c)** Axial T2 magnetic resonance imaging (MRI), **(d)** gradient echo MRI. MR images of right frontal superficial cavernous malformation.

issue here is that these extremely sensitive images can detect brain microhemorrhages that are basically the same as tiny CMs, so consideration needs to be given to what the etiology of any other abnormalities is. When operating in or near eloquent cortex, either or both functional MRI, diffusion tensor imaging and intraoperative electrophysiological monitoring are highly recommended. Awake craniotomy is another option for these cases. Frameless stereotactic image acquisition for preoperative craniotomy planning and intraoperative navigation may be considered mandatory for localization. Ultrasound should also be prepared for intraoperative use to help locate small subcortical lesions. General neurosurgical principles apply for all craniotomies performed for the removal of SCMs, including the administration of perioperative antibiotics. Consideration should be given to anticonvulsant usage in patients not already on these drugs. When the indication for surgery is medically refractory seizures, consultation with a seizure specialist should be considered to accurately assess the likelihood of obtaining improvement in seizure control when surgical resection is being considered. Removal of an SCM for amelioration of seizures should include the additional goal of excising the surrounding hemosiderin-stained tissue to decrease the relative excitability of the surrounding cortex that can contribute to seizure formation. The location of any associated developmental venous anomaly should be determined since this venous drainage should not be injured.

28.4 Operative Procedure

The patient is placed under general anesthesia and the head is placed in a pin fixation device. The patient's images are registered using frameless stereotaxis for craniotomy flap planning. The positioning and incision will be determined by the location of the lesion (▶ Fig. 28.3). The scalp is then prepared and draped in a standard manner. The skin incision is made and scalp hemostasis maintained with Raney hemostatic clips. The scalp flap is reflected with fishhooks and a standard craniotomy is performed using a high-speed drill with one burr hole, or multiple burr holes if the dura is likely to be adherent to the bone or the craniotomy crosses a venous sinus. Blunt dissection of the underlying dura mater is performed using a Love–Adson dissector (Codman, Johnson & Johnson), and the bone flap is removed using the high-speed drill. Hemostasis following bone flap removal is controlled with bone wax and microfibrillar collagen. The high-speed drill is used to create holes circumferentially around the bone flap for dural tackup sutures. The dura is opened in a cruciate or curvilinear fashion and retracted using

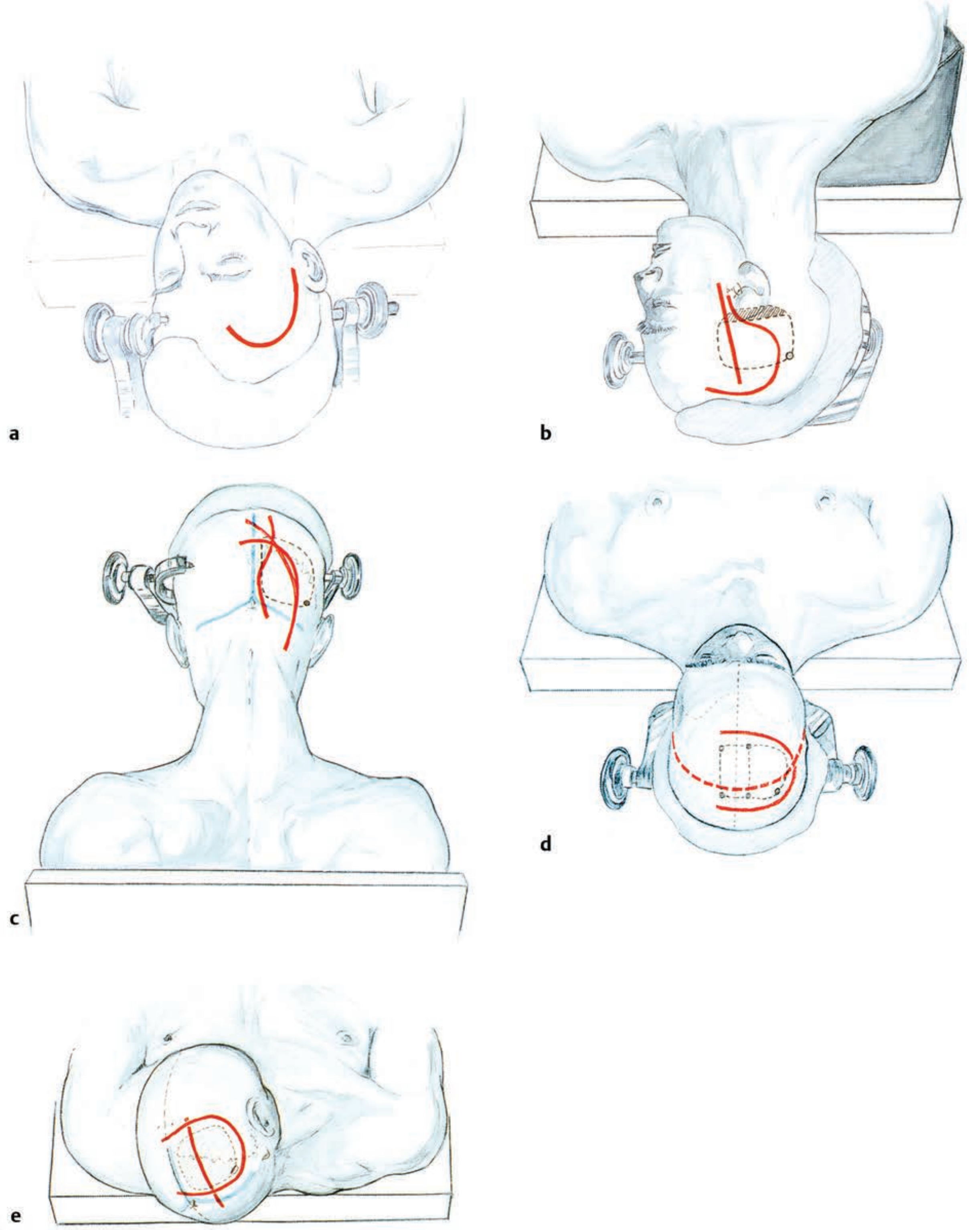

Fig. 28.3 Positioning and incisions for approaches to superficial CMs. **(a)** Pterional approach for lesions in the vicinity of the anterior sylvian fissure. **(b)** Approach to the middle cranial fossa showing two types of incisions and the bone removal, including removal of bone down to the floor of the middle fossa (*shaded area*) necessary to approach lesions on the undersurface of the temporal lobe. This exposure would also access lateral temporal lobe CMs. **(c)** Occipital paramedian approach with various incisions and a bone flap to access the occipital lobe. **(d)** Approach to lesions over the frontoparietal convexity near the midline. **(e)** Frontal midline approach to the medial frontal lobe. (Reproduced with permission from Mohsenipour I, Fischer J, Platzer W, Pomaroli A, eds. Approaches in Neurosurgery: Central and Peripheral Nervous System. New York: Thieme; 1994. Pp. 49, 56, 66, 74, 78.)

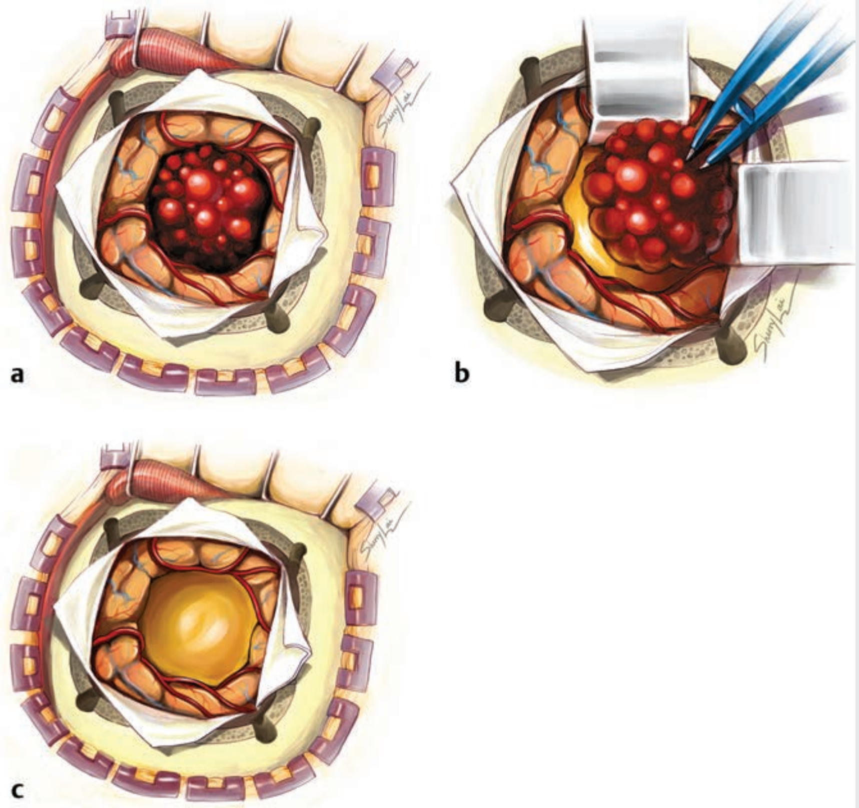

Fig. 28.4 **(a)** Initial intraoperative exposure of a superficial cavernous malformation. **(b)** The lesion has been excised completely, **(c)** leaving the hemosiderin-stained brain visible around it.

4–0 braided nonabsorbable suture held by hemostatic clips or by suturing to the scalp flap.

Frameless stereotaxis is used to localize the lesion. This is especially helpful if no hemosiderin-stained pia can be seen. Intraoperative ultrasound can be used to confirm the location of the lesion in real time, if required. Surgical excision of SCMs is relatively straightforward. Frequently the most challenging component of the operation is the localization of a small CM.

The microscope is brought in, and two microsurgical techniques generally used are the transsulcal and the transgyral approach. The former opens the arachnoid of a sulcus over the lesion, separates the pial surfaces, and then incises the cortex at the depth of the sulcus right over the lesion. Once localized, the CM will appear as a well-circumscribed, bluish purple, tabulated mass that looks like a mulberry (▶ Fig. 28.4, ▶ Fig. 28.5, ▶ Fig. 28.6). They lack a true capsule but are usually surrounded by a well-defined, firm, gliotic, hemosiderin-stained plane. Bleeding is rarely a problem because they do not have any major arterial supply. Associated developmental venous anomalies, however, can bleed if injured, but these should be preserved because they drain normal surrounding brain.

The well-defined gliotic plane allows for relatively easy dissection of the lesion from the surrounding brain using a combination of bipolar cautery and gentle suction. The malformation can be grasped with biopsy forceps and removed en bloc or in a piecemeal fashion depending on its size. If there is a history of recurrent hemorrhage it can sometimes be difficult to delineate clot from malformation, making definitive complete resection challenging. In the context of seizure surgery, the hemosiderin gliotic plane should also be removed when safe, otherwise the gliotic, hemosiderin-stained brain can be left behind. Once the CM is excised the pial walls are irrigated and inspected for any residual CM, and once fully excised, the pial cavity can be lined with oxidized regenerated cellulose if desired.

The dura mater is then closed in a watertight fashion, and the bone flap is replaced and fixed with titanium plates and screws. The scalp flap is reapproximated using 3–0 absorbable sutures for the galea aponeurosis and surgical clips or a running subcuticular suture.

28.5 Postoperative Management Including Possible Complications

Standard postcraniotomy surgical issues apply following removal of an SCM. The most dreaded complication following removal of an SCM is a postoperative hemorrhage causing either a focal neurological deficit or a diminished level of consciousness. With complete removal of the SCM this is an uncommon occurrence; however, with incomplete resection of the lesion this may lead to an increased risk of such an event. Exacerbation of a seizure disorder requiring aggressive anticonvulsant therapy is also possible in the postoperative period. Postoperative MRI done some months after surgery when postoperative enhancement has resolved is indicated to ensure the lesion has been completely resected.

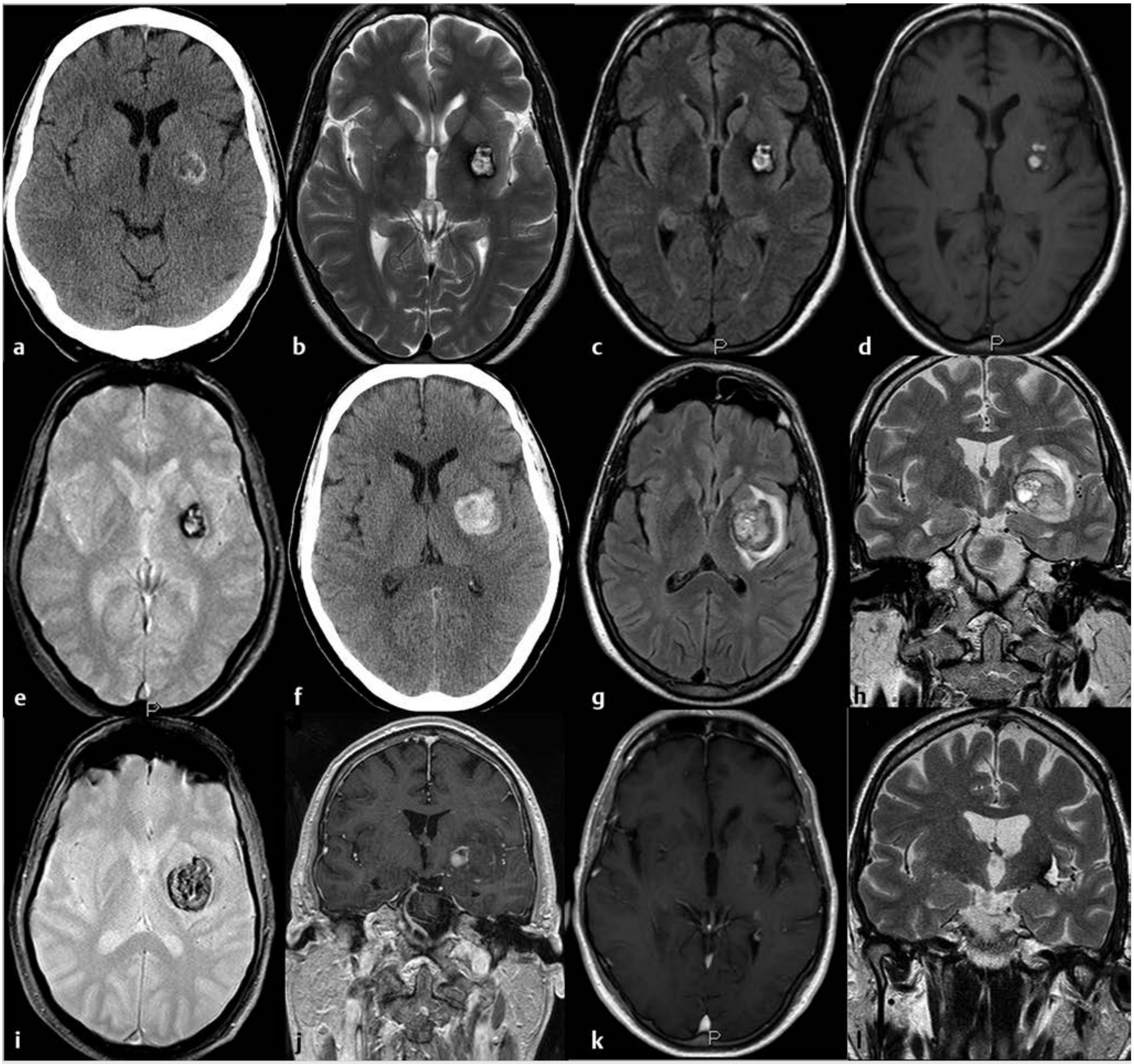

Fig. 28.5 Axial computed tomography (CT) **(a)** and magnetic resonance imaging (MRI) (T2 [**b**], FLAIR [**c**], T1 [**d**] and T2 enhanced steady-state gradient echo [**e**]) images of a 54-year-old right handed man with a CM in the left hemisphere deep to the insular cortex. He had had one prior hemorrhage associated with dysphasia and right weakness. The symptoms and signs resolved and he returned to work. Eight months after these images, he developed dysphasia and right lower limb weakness. Axial CT **(f)** shows acute hemorrhage at the location of the CM. A MRI with axial FLAIR **(g)** and gradient echo **(i)** and coronal T2 **(h)** and gadolinium-enhanced T1 **(j)** images show there has been hemorrhage from the CM. Diffusion tensor imaging showed the corticospinal tract was medial to the lesion. Surgical resection was undertaken 2 months later. The lesion was completely resected as determined by MRI 2 years later (axial T1 gadolinium enhanced [**k**] and coronal T2 [**l**] images). The patient had mild right distal lower extremity weakness. He returned to work full time and was well at his last follow-up 9 years later.

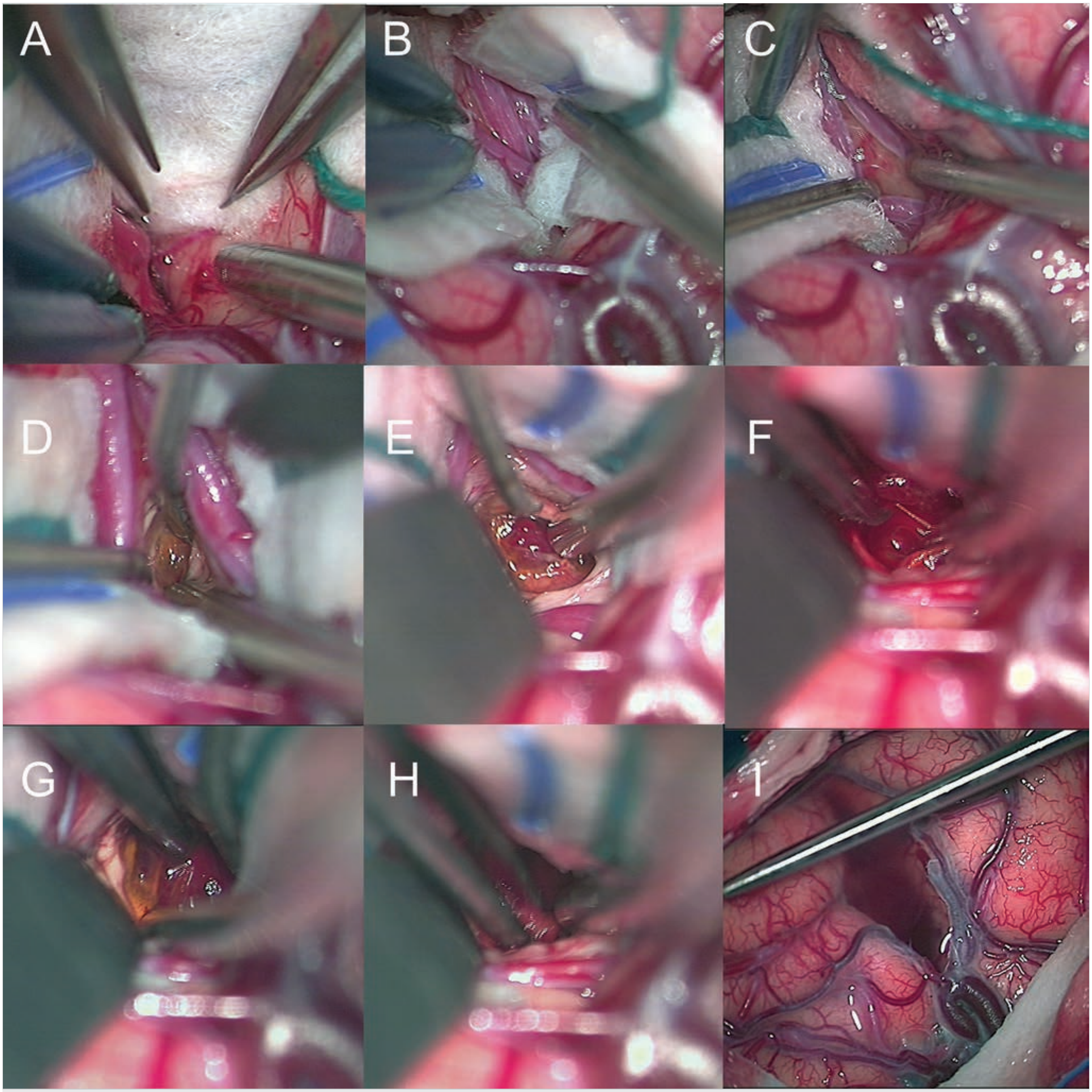

Fig. 28.6 Initial surgical exposure and opening of the sylvian fissure **(a)**. The frontal lobe is on the left and the temporal lobe on the right and the sylvian fissure courses obliquely across the photograph from about 5 o'clock up to 11 o'clock. The sylvian fissure is opened and second order branches of the middle cerebral artery exposed **(b)**. These are separated showing hemosiderin-stained insular cortex **(c)**. The cortex is opened and millimeters below that gliotic, hemosiderin-stained white matter is exposed **(d)**. The CM is exposed **(e)** and some of the mulberry-like saccules of the lesion are seen **(f)**. Progressive circumferential dissection between the CM and the hemosiderin-stained brain **(g)** is done and the lesion is grasped with cup forceps and removed **(h)**. The brain is uninjured at the end of the removal **(i)**.

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29 Brainstem Cavernous Malformations

Hussam Abou-Al-Shaar, Mohamed A. Labib, and Robert F. Spetzler

Abstract
Brainstem cavernous malformations are uncommon vascular malformations of the central nervous system. These lesions frequently pose a management challenge to neurosurgeons. Brainstem cavernous malformations can be addressed with different management paradigms, including observation, radiotherapy, and surgical resection. Observation is considered acceptable for small incidentally found lesions, whereas the beneficial effects of radiotherapy are still controversial, and many authors recommend against its use. Surgical resection during the subacute stage can achieve high cure rates with minimal, if any, morbidity. Optimal selection of the surgical approach results from detailed knowledge of the safe entry zones to the brainstem. However, it is essential to educate patients about the risks and benefits of surgery and the possible complications.

Keywords: brainstem, cavernoma, cavernous malformation, intraoperative monitoring, microsurgical resection, natural history, safe entry zones, surgical anatomy, surgical approaches, two-point method

29.1 Epidemiology

Cavernous malformations (CMs) constitute 5 to 13% of all vascular lesions in the brain and spinal cord.[1] With advanced imaging techniques, the detection rate, and therefore the incidence, of these lesions appears to have increased over the past three decades. The incidence of CMs is estimated to be 0.4 to 0.5% among the general population, and the prevalence is estimated to be 0.4 to 0.8%.[2,3,4]

CMs are vascular malformations consisting of endothelium-lined, grossly dilated, vascular channels that lack the typical tight junctions of normal blood vessels. The absence of tight junctions allows for blood to extravasate from the malformed vessels.[5] CMs can occur in any location in the central nervous system (CNS). They are mostly supratentorial; however, infratentorial CMs are well described. They occur less frequently in the brainstem. An estimated 15 to 18% of all CNS CMs arise in the brainstem.[6] The majority of brainstem CMs occur in the pons, followed by the midbrain and finally the medulla. This distribution has been attributed to the sheer volume of tissue in the pons compared to that in the other segments of the brainstem.[7,8]

29.2 Clinical Presentation

The clinical presentation of patients with brainstem CMs depends on the size and location of the lesion. Patients with these lesions can be completely asymptomatic or can die as a result of the CM. The most common reported presentation is cranial neuropathy. Other manifestations include sensory loss, motor deficits, ataxia, headache, nausea, vertigo, and dysarthria. Recognition of these distinct clinical features can aid the neurosurgeon in localizing the malformation within the brainstem.[7]

29.3 Imaging Characteristics

Magnetic resonance imaging (MRI) is the most sensitive and specific imaging modality available for diagnosis of CMs. These lesions are hypointense or isointense on T1-weighted MRI, whereas they have a heterogeneous “popcorn” appearance with mixed hyperintense and hypointense signals on T2-weighted MRI. Their hypointense appearance on gradient echo T2* images (due to hemosiderin deposition in and around the CM) is virtually pathognomonic. CMs generally do not enhance upon administration of intravenous gadolinium. The role of computed tomography is limited for the diagnosis of brainstem CMs. Typically, such lesions have a hyperdense appearance with or without calcification. CMs are angiographically occult because of low or absent blood flow and the high incidence of thrombosis.[9,10,11] Nonetheless, a catheter angiogram may be helpful in excluding a small arteriovenous malformation in patients with acute hemorrhage when other diagnostic modalities have not been definitive.

29.4 Natural History

The pathogenesis of CMs remains largely unknown. They are thought to arise during early embryogenesis and then to grow according to changes in blood and mechanisms of malformation.[12] However, many CMs arise de novo, with some occurring after radiotherapy.[13] Most lesions are solitary and sporadic, whereas some are multiple and inherited in an autosomal dominant fashion.[14]

The natural history of CMs, particularly those present since birth, stems from their risk of bleeding and rebleeding. This topic has been an area of debate for the past few decades. Retrospective reports indicate an annual risk of hemorrhage of 0.5 to 2.7% and of rehemorrhage of 21 to 60% per year; prospective studies report a hemorrhage rate of 0.2 to 0.7% and a rehemorrhage rate of 5 to 7% per year.[3,7,15,16,17,18,19] The hemorrhage and rehemorrhage rates for brainstem CMs (especially during the first 2 years) have been estimated to be higher than the rates for CMs in other locations.[16,20,21] Moreover, although numerous CM hemorrhages go unnoticed, the majority of brainstem CM hemorrhages produce symptomatic clinical features because of the eloquence of this region. Studies have demonstrated a significant increase in the risk of rehemorrhage among patients with a previous CM hemorrhage.[15,19,22]

Garcia and colleagues developed a grading system to categorize brainstem CMs that is similar to the widely used Spetzler–Martin grading system for brain arteriovenous malformations.[7,23] In their grading system, five elements (age, lesion size, extension across the midline, the presence of an associated developmental venous anomaly, and the presence of hemorrhage) are graded for a total score of up to seven points. The total score is used to assign a grade (from 0 to 7) to the patient, which predicts the surgical morbidity and outcome (higher total score associated with a worse outcome).

29.5 Management Options

The optimal management of brainstem CMs is widely debated. Three management paradigms constitute the most commonly used modalities to address these lesions.

29.5.1 Observation

Conservative management of brainstem CMs in the form of clinical observation and neuroimaging surveillance is acceptable both for patients with incidentally discovered asymptomatic CMs and for patients with complete clinical recovery after an initial hemorrhage. The risk of hemorrhage among incidentally discovered brainstem CMs has been reported to be less than 1% per year.[7,15,16,17,18,19] The higher rates of morbidity (9.6–45%) and mortality (0.96–1.5%) associated with surgical intervention make conservative management of such patients preferable.[6,7,24,25]

29.5.2 Radiotherapy

The role of radiotherapy, including in its various modes of delivery (stereotactic radiotherapy or gamma knife [Elekta AB] radiotherapy), has produced contradictory results for the management of brainstem CMs. Radiosurgery has been recommended specifically for CMs arising in locations not amenable to surgical intervention. However, this modality is also associated with high rates of morbidity (15–59%) and mortality (0–8%).[26,27,28,29]

Although some reports document the superiority of radiotherapy compared to conservative management, others have argued that the observed improvement among patients with brainstem CMs is merely due to the natural history of the disease rather than the result of the radiotherapy.[27,30,31] Radiotherapy has even been implicated as a culprit in the pathogenesis of these lesions.[13] Notably, the decrease in the hemorrhage rate observed in patients with lesions treated with radiotherapy occurs mainly after a latent period of 2 years.[32] Similarly, CMs that are managed conservatively have a substantially decreased risk of spontaneous hemorrhage after 2 years. Thus, the question remains regarding whether the observed hemorrhage reduction is due to radiotherapy or the natural history of CMs.[31] Moreover, as Almefty and Spetzler pointed out, radiologic studies obtained after CMs were treated with radiotherapy have failed to demonstrate resolution of the CMs, further questioning the clinical efficacy of this modality.[31] Thus, there is inadequate evidence-based medicine to document the superiority of radiotherapy over conservative management or vice versa.

29.5.3 Surgery

For decades, surgery has been contraindicated in the brainstem, which has been considered to be a "no man's land." The presence of multiple cranial nerve nuclei and tracts in this small area make any manipulation a great risk for morbidity and mortality.

Surgical intervention generally depends both on the location of the lesion within the brainstem and on the clinical presentation of the patient. Indications for surgery of brainstem CMs include, but are not limited to, a lesion abutting the pial surface of the brainstem, bleeding with progressive neurologic deterioration, acute hemorrhage outside the capsule of the lesion, and clinically significant mass effect caused by intralesional bleeding.

Hemorrhages of brainstem CMs are generally classified as acute, subacute, or chronic.[7,24,33] During the acute stage, surgical intervention should be avoided because of the firm nature of the hematoma and the presence of extensive edema, which limits the degree of manipulation and exploration of the lesion. During the subacute stage of hemorrhage (6–8 weeks after the initial hemorrhage), the edema resolves and the blood liquifies. This process creates a surgical plane that separates the lesion from the surrounding parenchyma, which aids the neurosurgeon in establishing complete resection of the lesion. Finally, during the third stage of chronic hemorrhage, extensive fibrosis, gliosis, and hematoma reorganization ensue. This process causes the CM to adhere firmly to the surrounding eloquent parenchyma, which limits the exposure and the ability to manipulate microsurgical instruments.[7,24,33] In a retrospective study on the impact of the timing of surgical intervention for 397 patients with brainstem CMs, Zaidi et al, found that surgery within 6 to 8 weeks of hemorrhage was associated with a statistically significant likelihood of superior recovery compared to that of patients who undergo later surgery (odds ratio 1.73, 95% confidence interval 1.06–2.83, $p = 0.03$).[24] Therefore, most authors advocate surgical intervention during the subacute stage.

The presence of multiple CMs is considered a contraindication for surgical intervention unless the clinical manifestations can be attributed to a single lesion that might be addressed surgically. The outcomes of surgical intervention are delineated in the section on clinical outcomes.

29.6 Operative Considerations

The goal of surgery for brainstem CMs is to achieve complete surgical resection to prevent the risk of hemorrhage while minimizing the need to tranverse eloquent parenchyma. Subtotal resection has been shown to result in repeat hemorrhage rates of up to 62%.[6]

Some authors recommend opening the pial surface of the brainstem with microforceps instead of a blade so as to spread the neural fibers at the desired safe entry zone rather than cutting through them (▶ Table 29.1).[34] Moreover, it is not uncommon to see an associated venous anomaly in conjunction with a CM, and these must be preserved to avoid the risk of venous infarction.

The surgical approach depends on the location and size of the lesion, as well as on the expertise of the neurosurgeon. For the sake of simplification, a two-point method has been developed to determine the best surgical approach to reach the CM.[35] Two points are drawn on an MRI, one at the center of the lesion, and the other at the periphery closest to the pial surface. When the two points are connected, the resultant straight line determines the best surgical approach. Although this simplified method can be used in the majority of cases, some cases require a different approach than the one inferred by the two-point method because of the potential traversing of critical eloquent neural structures and pathways. Thus, knowledge of the brainstem anatomy and surgical approaches is essential in the surgical

Table 29.1 Safe entry zones to the brainstem

Approach	Safe entry zone
Orbitozygomatic	Anterior mesencephalic zone
Subtemporal	Anterior mesencephalic zone
Subtemporal transtentorial	Anterior mesencephalic zone and supratrigeminal zone
Anterior petrosectomy (Kawase)	Anterior mesencephalic zone, supratrigeminal zone, and peritrigeminal zone
Suboccipital ± telovelar	Median sulcus of fourth ventricle and superior fovea
Median supracerebellar infratentorial	Lateral mesencephalic sulcus, intercollicular zone, supracollicular zone, and infracollicular zone
Lateral/extreme lateral supracerebellar infratentorial	Lateral mesencephalic sulcus, intercollicular zone, supracollicular zone, and infracollicular zone
Retrosigmoid	Lateral mesencephalic sulcus, supratrigeminal zone, peritrigeminal zone, lateral pontine zone, anterolateral sulcus of medulla, posterior median sulcus of medulla, and lateral medullary zone
Far-lateral	Anterolateral sulcus of medulla, posterior median sulcus of medulla, lateral medullary zone, and olivary zone
Retrolabyrinthine	Lateral mesencephalic sulcus, supratrigeminal zone, peritrigeminal zone, lateral pontine zone, anterolateral sulcus of medulla, posterior median sulcus of medulla, lateral medullary zone, and olivary zone

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planning for treatment of these lesions (▶ Table 29.1, ▶ Table 29.2). We advocate the use of diffusion tensor imaging MRI when the direction of displacement of important white matter tracts is questionable.

The development of lighted microsurgical instruments has greatly aided surgical resection of brainstem CMs. These instruments function in a dual fashion, performing their intended task (e.g., suction) while also providing illumination to aid in the visualization of the lesion and the adjacent anatomy.[34] Proper patient positioning also allows the neurosurgeon to operate without having to use fixed retraction of eloquent brain tissue.[36]

The use of an intraoperative image-guided system integrated into the operative microscope is useful for the resection of brainstem CMs. It allows for optimal planning of, and navigation through, the surgical corridor.[37] Of equal importance is the use of intraoperative neurophysiological monitoring. Monitoring somatosensory evoked potentials, motor evoked potentials, electroencephalography, brainstem auditory evoked potentials, and cranial nerve function should be conducted during the surgical resection of brainstem CMs. As imaging techniques have advanced, intraoperative MRI has increasingly been proposed as a valuable adjunct in the management of brainstem CMs to ensure the complete removal of these lesions.[34]

Table 29.2 Surgical approaches for brainstem CMs

CM location	Approach
Midbrain	
Anterior	Pterional ± orbitozygomatic
Posterior	Median supracerebellar infratentorial
Anterolateral	Pterional ± orbitozygomatic
Posterolateral	Paramedian or extreme lateral supracerebellar infratentorial
Pons	
Anterior	Pterional ± orbitozygomatic, subtemporal ± transtentorial, retrolabyrinthine, and retrosigmoid
Posterior	Suboccipital ± telovelar
Lateral	Retrosigmoid
Medulla	
Anterior	Far-lateral and retrosigmoid
Posterior	Suboccipital ± telovelar
Upper lateral	Far-lateral and retrosigmoid
Lower lateral	Far-lateral

Abbreviation: CM, cavernous malformation. Data compiled from Kalani et al and Cavalcanti et al.[38,39]

29.7 Operative Procedure

Various surgical approaches are used for the management of brainstem CMs, depending on their location within the brainstem (▶ Table 29.2). As an example, a 56-year-old man presented with diplopia on right gaze. His examination revealed left upper limb weakness (4/5) and bilateral dysmetria. His imaging showed a midbrain CM (▶ Fig. 29.1). There was a history of three prior hemorrhages. A left lateral supracerebellar infratentorial approach was used to approach the lesion in the prone position (▶ Fig. 29.2, **Video 29.1**). Somatosensory evoked potentials and motor evoked potentials were monitored. Complete resection was achieved (▶ Fig. 29.3).

Detailed knowledge of these approaches, along with knowledge of the safe entry zones (▶ Table 29.2) to brainstem lesions that do not abut the pial surface, is essential to achieving high success rates with minimal morbidity (▶ Table 29.1). Nonetheless, there will always be some lesions that are not amenable to surgical intervention, regardless of the approach, because surgical intervention would be more harmful than the natural history of the disease itself.

29.8 Postoperative Management and Outcome

During the postoperative period, patients might have to remain intubated for 24 hours until they demonstrate a good cough and gag reflex. Swallowing should be assessed, especially among patients with medullary CMs. Neuroimaging should be performed on postoperative day 1 and annually thereafter to document complete removal of the malformation and the

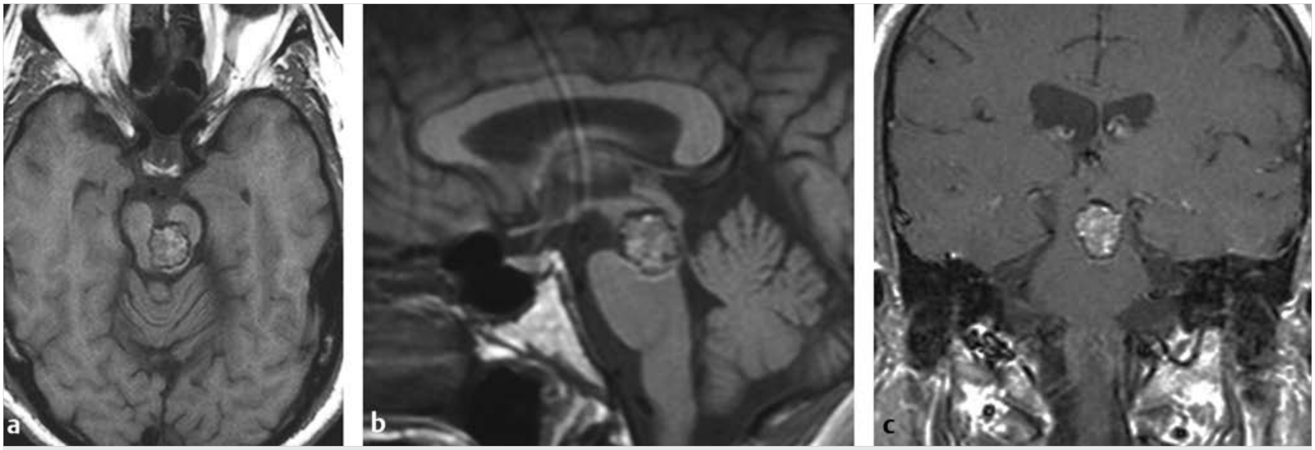

Fig. 29.1 Preoperative **(a)** axial, **(b)**, sagittal, and **(c)** coronal T1-weighted magnetic resonance images demonstrate a cavernous malformation in the midbrain of a patient with a history of three prior hemorrhages. (Used with permission from Barrow Neurological Institute, Phoenix, AZ.)

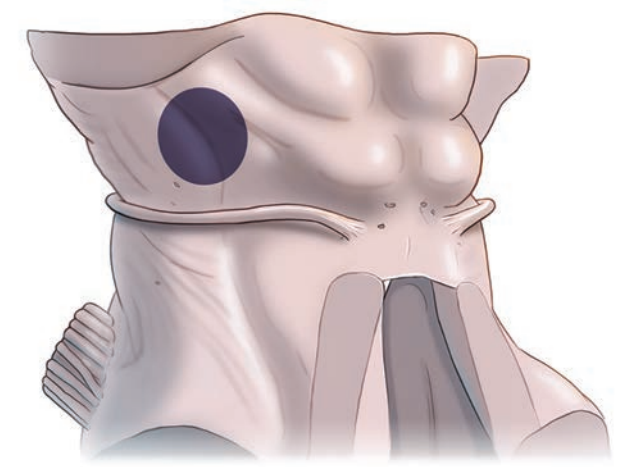

Fig. 29.2 Artist's illustration demonstrates the location (*filled circle*) of a midbrain cavernous malformation in a 56-year-old man who presented with diplopia on right gaze. (Used with permission from Barrow Neurological Institute, Phoenix, AZ.)

absence of any residual tissue. Finally, patients should be advised that recurrence can occur in up to 5% of cases, even in the absence of radiologic evidence of residual tissue.

The clinical outcomes of brainstem surgery for CMs are generally favorable. In our series of 100 patients with 103 brainstem CMs, 86 underwent surgical resection.[16] At a mean follow-up of 35 months available for 84 of the 86 surgical patients, 87% ($n = 73$) had improved or were unchanged, 10% ($n = 8$) had deteriorated, and 4% ($n = 3$) had died. However, among the 12 of 14 patients who did not receive surgical intervention and were available for follow-up, 58% ($n = 7$) had improved or had remained the same, 42% ($n = 4$) had deteriorated, and 8% ($n = 1$) had died. Dukatz and colleagues reported a similar experience with 71 patients with CMs who were treated surgically.[40] In their series, 63% ($n = 44$) of patients demonstrated improvement in Karnofsky Performance Scale status, 27% ($n = 19$) reported no change, and 11% ($n = 8$) showed deterioration. Garcia et al, reported good outcomes in 79.8% ($n = 83$) of 104 surgically treated patients, in whom the complication rate was 20.2% ($n = 21$).[7]

It is common for patients with CMs who undergo resection to experience transient postoperative deficits or long-term neurologic deficits. Abla et al, reported new deficits or a worsening of preoperative deficits in their long-term study of 260 patients with brainstem CMs that were managed surgically.[25] Of the 240 patients available at a mean follow-up of 51 months, 53% ($n = 137$) were worse after surgery, whereas 36% ($n = 93$) had developed new permanent deficits.

To better understand the efficacy of surgical intervention in this patient population, Gross et al, conducted a meta-analysis of 68 surgical series, reviewing 1,390 cases of brainstem CMs.[6] These authors found that in 61 reports, 91% (1,178 of 1,291) of the patients had complete resection and that in 60 reports, 84% (889 of 1058) were improved or the same, whereas 62% (65 of 105) with partial resection had rehemorrhaged. Almost one-half (45%; 425 of 944 patients in 46 series) had early neurologic morbidity, and 1.5% (21 of 1390 patients in all 68 series) had died. However, 62% (609 of 987) of the patients across 51 series had improvement of their symptoms over the follow-up period.

These results support our conviction that surgical intervention for brainstem CMs, when indicated, is a safe and effective cure for such lesions. In experienced hands and with appropriate treatment selection, a high cure rate can be achieved with minimal, if any, morbidity.

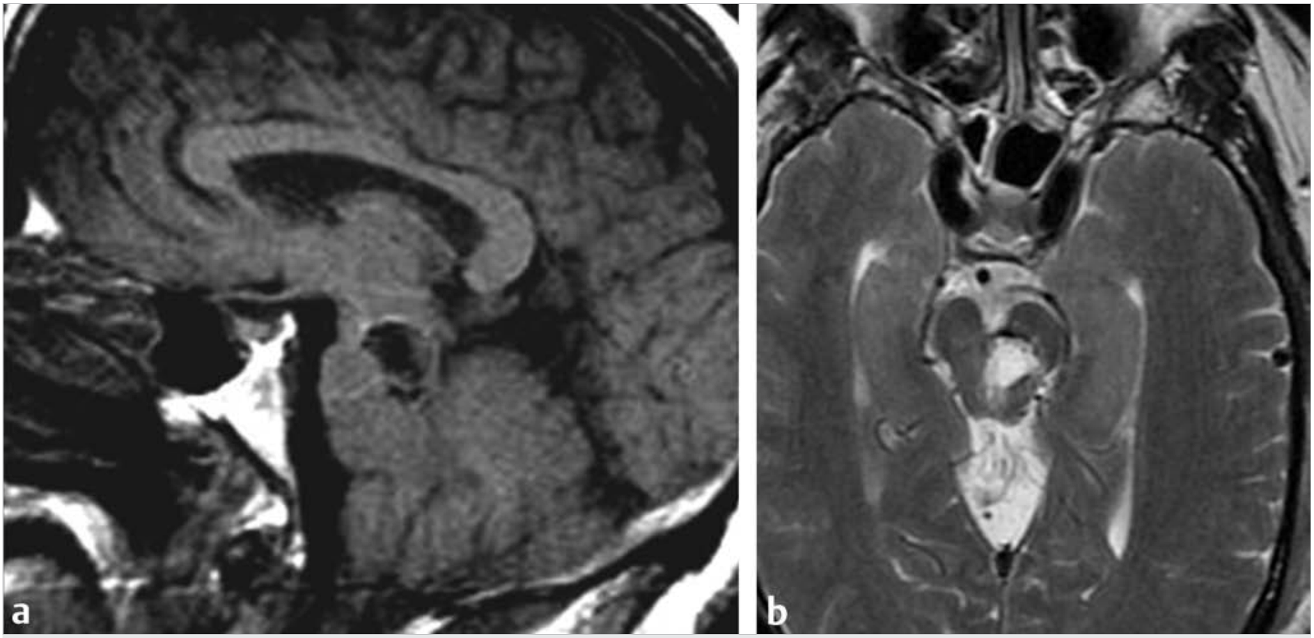

Fig. 29.3 Postoperative **(a)** sagittal T1-weighted and **(b)** axial T2-weighted magnetic resonance images demonstrate complete removal of the cavernous malformation. (Used with permission from Barrow Neurological Institute, Phoenix, AZ.)

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30 Spinal Vascular Malformations

R. Webster Crowley and Edward H. Oldfield

Abstract
Spinal vascular malformations include a spectrum of lesions that range from simple to treat with low risk such as dural arteriovenous fistulas, to complex, inoperable lesions. Most intramedullary vascular malformations, even when potentially curable, carry the possibility of devastating complications such as paraplegia or quadriplegia. This chapter reviews the different spinal vascular malformations and provides a framework for understanding their clinical characteristics and treatment options.

Keywords: arteriovenous malformation, spinal cord vascular malformation, dural arteriovenous fistula

30.1 Patient Selection

Treatment of patients with spinal arteriovenous malformations (AVMs) carries a risk of paraplegia or quadriplegia. Therefore, before treatment is recommended, the natural history of the lesion must be weighed against the risks of therapy. Once neurological symptoms appear, most patients with dural arteriovenous fistulas (AVFs) become progressively disabled, and many are confined to bed or a wheelchair within months. The risk of treatment is low so these patients should be treated promptly. In contrast, the intradural AVMs, AVFs, and cavernous malformations have a less predictable natural history, the risk of treatment is higher, and there is less likelihood of complete obliteration of the lesion. Therefore, treatment decisions need to be individualized in these cases. If the patient has hemorrhaged, usually treatment is delayed until the hemorrhage resolves.

Successful treatment requires: (1) diagnosis of the AVM before irreversible cord injury occurs; (2) determination of the site and the type of AVM and delineation of the vascular anatomy of the AVM and of the vessels supplying the spinal cord in the vicinity of the AVM; (3) coupling the goals of surgery with the mechanism of cord injury, which differs in the different types of spinal vascular malformations; and (4) successful execution of the planned surgery. The goal of treatment is safe, complete, permanent obliteration of the lesion.

30.1.1 Classification of Spinal Vascular Malformations

There are three main types of vascular abnormalities of the spinal cord. Dural AVFs are embedded in the dura of the proximal portion of the nerve root sleeve and adjacent spinal dura. Intradural AVMs have a nidus in the spinal cord or on the surface of it and are further categorized into AVMs of the spinal cord, juvenile and glomus AVMs, and pial AVFs, in which a direct arterial to venous transition occurs (usually on the surface of the spinal cord) without an intervening glomus of abnormal vessels. Cavernous malformations of the spine are located within the parenchyma of the spinal cord.

30.2 Preoperative Preparation

A spinal catheter angiogram is generally done for all spinal AVMs and AVFs. In the past, patients with suspected spinal vascular lesions often underwent exploratory angiograms of the entire spine to locate the lesion. However, recent improvements in magnetic resonance angiography and computed tomography angiography have largely obviated the need for full spinal angiograms, as they are quite effective at identifying vascular lesions, as well as the specific level and side of associated feeding arteries. A formal arteriogram is still recommended; however, in combination with available noninvasive imaging, the arteriogram can be targeted to the suspected levels. This greatly reduces the patient's exposure to radiation and contrast. Imaging at a rapid rate (6–10 frames/s) is recommended. With the intradural AVMs and AVFs, preoperative embolization performed during arteriography 1 to 2 days before surgery reduces the blood flow through the lesion and facilitates manipulation of the vessels during surgery. High-dose glucocorticoid therapy is administered just before, during, and for 24 to 48 hours after surgery or embolization. Intravenous heparin is administered for 48 hours when embolic occlusion is complete.

30.3 Operative Procedure

30.3.1 Dural Arteriovenous Fistulas

The options for treatment include surgery to interrupt the vein draining the AVF as it penetrates the inner layer of dura (▶ Fig. 30.1**,** ▶ Fig. 30.2**,** ▶ Fig. 30.3**, Video 30.1**) or endovascular embolization. Embolic occlusion with particulate materials, such as polyvinyl alcohol, does not permanently obliterate the nidus and therefore is typically not used for fistulas. Conversely, embolization with polymerizing liquid embolic agents such as Onyx or n-butyl cyanoacrylate may provide a durable occlusion, and an increasing number of centers are now preferentially treating dural AVFs using endovascular methods. Just as the goal of surgical ligation is to interrupt the draining vein as it enters the dura, an endovascular cure requires penetration of the embolysate to the point of fistula, and often into the venous side of the fistula. This is not always safe or feasible due to a number of factors such as the tortuosity of the arterial supply to the fistula, or the involvement or proximity of medullary arteries that supply the normal spinal cord such as the Artery of Adamkiewicz. Even in cases where embolization is felt to be a possibility, the embolysate may not penetrate to the fistula, which typically results in an incomplete occlusion. Lastly, recanalization of a presumably cured fistula has been reported. Surgery, on the other hand, is often fairly straightforward, and most of these factors that make embolization difficult do not impact surgical treatment. For these reasons, a substantial percentage of spinal DAVFs remain best managed surgically.

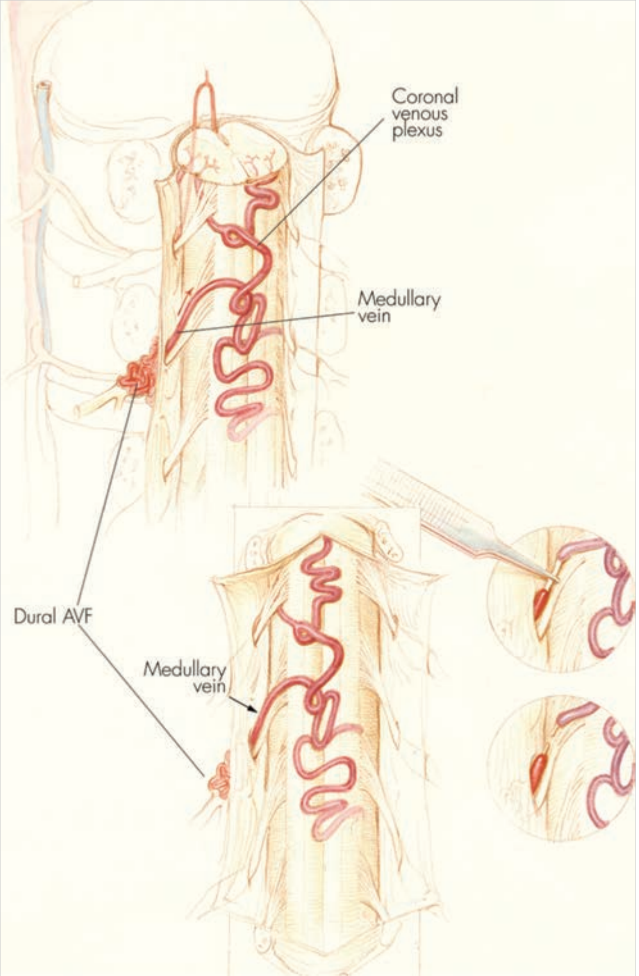

Fig. 30.1 The vascular anatomy of a spinal dural arteriovenous fistula (AVF). The AVF is supplied by a dural artery and drained by a medullary vein. Arterial input into the valveless intradural venous system increases venous pressure within the coronal venous plexus and causes myelopathy. Treatment is to coagulate and divide the arterialized draining vein at the site of the intradural penetration of the AVF.

Interruption of the vein that carries blood from the dural AVF to the coronal venous plexus cures almost all of these patients (▶ Fig. 30.1, ▶ Fig. 30.2, ▶ Fig. 30.3, **Video 30.1**). During the preoperative arteriogram a marker placed on the patient's back, or a detachable coil placed endovascularly within the segmental artery at the level of the fistula, can provide a useful radiographic landmark for localizing the AVF at surgery. The lamina of the neural arches, one level above and below the AVF, are removed widely. The dura is opened in the midline, the arachnoid is separated from the underlying vessels, and the dura and arachnoid are retracted laterally with dural sutures. Correlation of the vascular anatomy as seen on preoperative arteriography with that displayed intraoperatively permits identification of the site of dural penetration of the medullary vein carrying arterial blood to the dilated, tortuous coronal venous plexus on the surface of the spinal cord (▶ Fig. 30.2, ▶ Fig. 30.3, **Video 30.1**). Indocyanine green (ICG) microscopy is useful to confirm identification of the target vessel. This arterialized vein is then coagulated with bipolar forceps and sharply divided as it enters the dura. This simple procedure provides effective, permanent therapy for almost all patients.

30.3.2 Intradural Arteriovenous Malformations

The nidus of juvenile and glomus spinal AVMs involves the spinal cord (▶ Fig. 30.4, ▶ Fig. 30.5). One of the feeding vessels of these AVMs is almost always an enlarged medullary artery that also supplies the spinal cord. Because most of these AVMs occupy the ventral half of the spinal cord, they are usually supplied by the anterior spinal artery. Juvenile AVMs (▶ Fig. 30.4) usually become symptomatic in children and young adults. These are high-flow lesions like cerebral AVMs, with multiple feeding arteries supplying a voluminous AVM. They are not usually operated upon. In contrast, the glomus AVM (▶ Fig. 30.5) is a more localized nidus of smaller blood vessels confined to a short segment of spinal cord that generally affects adults. Flow varies from low to high depending on the patient.

Glomus AVMs, particularly the posterior lesions (▶ Fig. 30.5) or those in the cervical spinal cord, where collateral flow in the anterior spinal artery can arise inferiorly or superiorly, can occasionally be totally excised safely. However, surgical excision is hazardous with the more common ventral lesions in the thoracic and lumbar segments of the spinal cord, where collateral blood supply to the spinal cord is limited.

Operative exposure of intramedullary spinal AVMs is performed in the prone position with a complete laminectomy, covering at least one segment above and below the nidus of the malformation. The dura is opened in the midline, keeping the arachnoid intact in order to avoid tearing of the large, fragile underlying vessels (▶ Fig. 30.5). The arachnoid is opened separately and sutures retract the dura and arachnoid laterally. Dissection around the AVM with meticulous hemostasis is essential because blood-stained pia obscures the anatomical details. Controlled hypotension is useful with tightly distended AVMs. Liberal irrigation during bipolar coagulation prevents coagulated vessel walls from adhering to the tips of the forceps. Larger feeding vessels may require ligatures or clips, but most vessels can be managed with simple bipolar coagulation and interruption. A watertight dural closure is performed.

30.3.3 Intradural Perimedullary Arteriovenous Fistulas

These are simple AVFs in the pia (▶ Fig. 30.6) with one to three direct shunts between an artery and a vein, frequently with an aneurysm or venous varix at, or near, the transition from artery to vein. They are predominantly anterior lesions of the lower portion of the spinal cord where they are supplied by the anterior spinal artery, but they occasionally occupy a dorsolateral position, and when they do, they tend to be supplied predominantly by a posterior spinal artery (▶ Fig. 30.6, ▶ Fig. 30.7). They usually present with progressive myelopathy. Embolization can be curative in a certain percentage of these AVMs. Otherwise, surgical disconnection of the fistula is indicated. As is the case with the preceding vascular lesions, ICG microscopy can be useful in locating the exact point of fistula(s) during surgery.

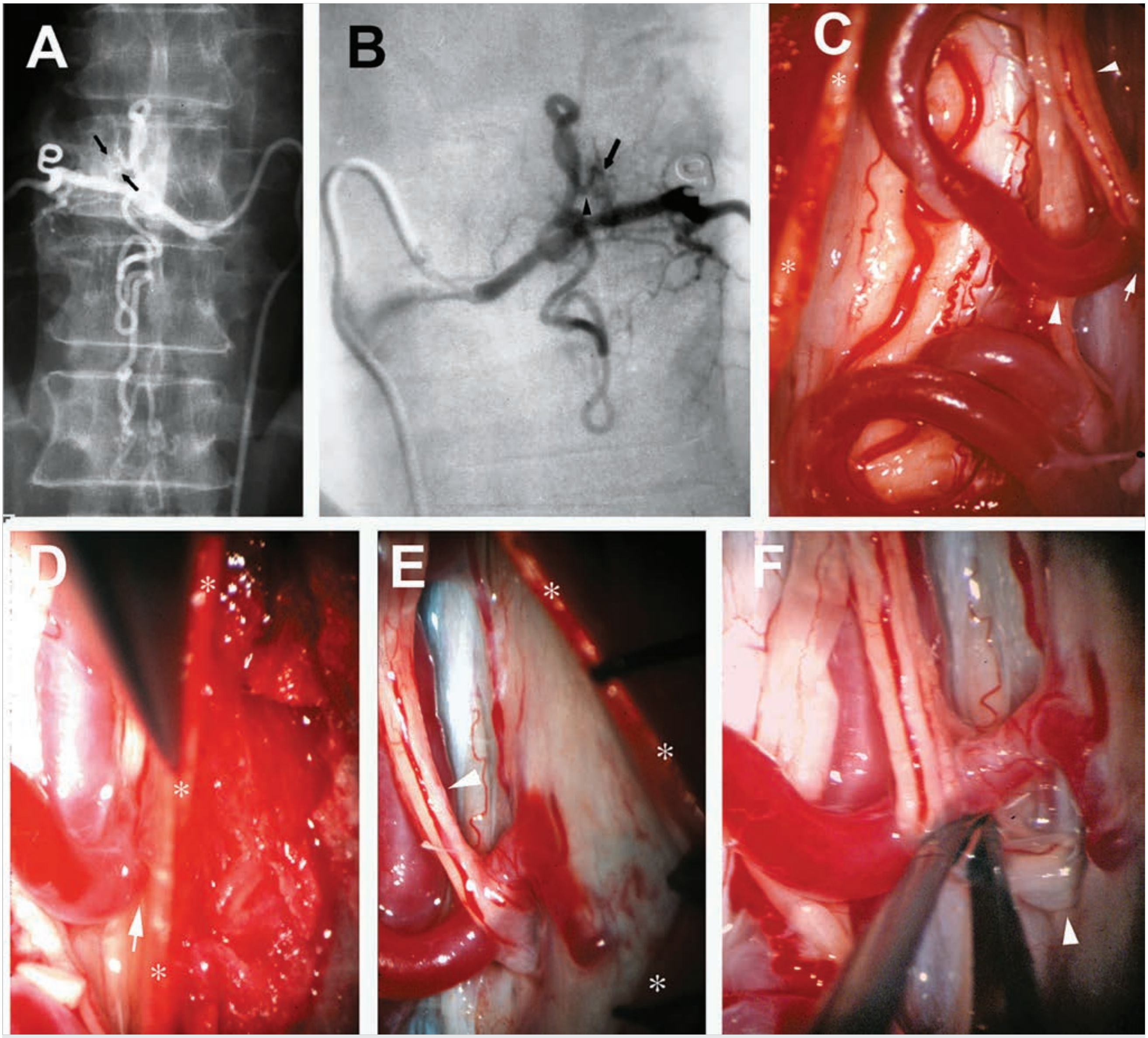

Fig. 30.2 **(a)** Selective spinal arteriogram of a spinal dural arteriovenous fistula (AVF) embedded in the root sleeve of the ninth right thoracic nerve root. The nidus of the AVF (*arrows*) is typically in the intervertebral foramen and the lateral aspect of the spinal canal and drains into dilated, tortuous intradural veins on the cord surface. **(b)** Subtraction arteriogram with the image reversed to correspond to **(c–f)** the view at surgery with the patient prone. The upward-pointing arrowheads in **(b)** and **(c)** indicate the caudal loop of the medullary vein draining the dural AVF. The left pointing arrowhead in **(c)** indicates the right T9 sensory root, and the arrow points to the site of dural penetration of the vein draining the AVF and the sensory root. In **(d)** the forceps grasp the dura (*asterisk*). This intra and extradural view shows the AVF imbedded in the dura (to the right of the dura in the image) and the site of intradural penetration of the medullary vein draining the AVF intradurally (*arrow*). In **(e)** and **(f)** the dura (*asterisk* in **e)** is retracted laterally, revealing the relationship of the nerve root and the dural penetration of the arterialized medullary vein that drains the blood from the fistula intradurally to the spinal venous system. (a, d, and e reproduced with permissions from Oldfield E, DiChiro G, Quindlen E, et al. Successful treatment of a group of spinal cord arteriovenous malformations by interruption of dural fistula. J Neurosurg 1983:59:1019.)

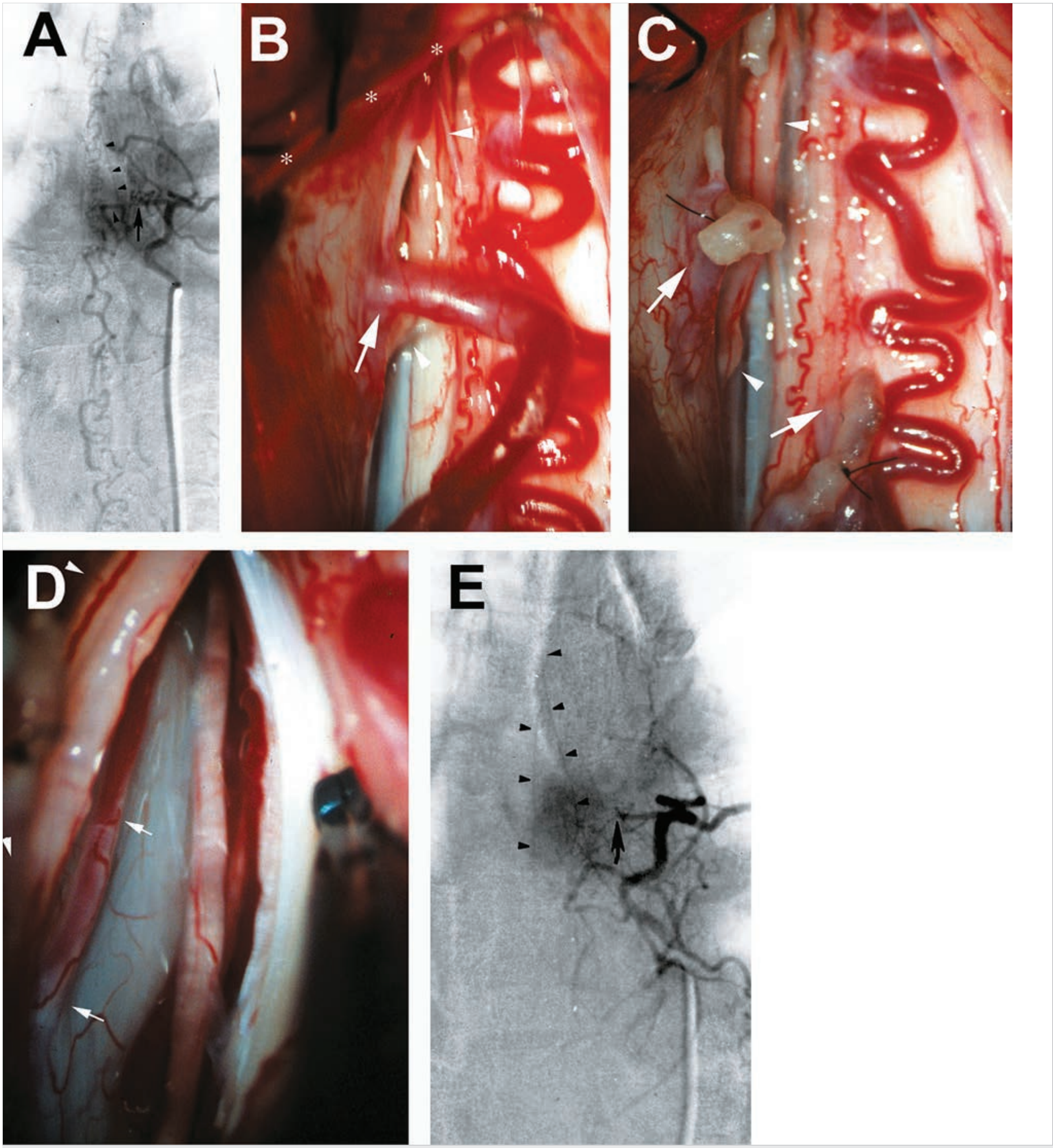

Fig. 30.3 Selective spinal arteriogram in a 36-year-old male with a spinal dural arteriovenous fistula (AVF) at the left seventh thoracic nerve root. **(a)** The seventh left thoracic intercostal artery provides a common origin of the arterial supply to the dural AVF (*arrow*) and the artery of Adamkiewicz (*arrowheads* pointing to the left of the image). Note the medullary vein draining the AVF intradurally (*arrowhead* pointing upward) takes a horizontal course initially. **(b–d)** Surgical view (patient prone) after the dura (*asterisk*) and arachnoid have been opened. **(b)** The medullary vein draining the AVF enters the dura *(arrow)* just dorsal to the left seventh thoracic sensory root (*arrowheads*). **(c)** Medullary vein draining the dural AVF has been coagulated and divided (*arrows*) as it enters the subdural space (*upper arrow*). Note the dural penetration of the sensory root just deep to the site of intradural entry of the vein (*lower arrowhead*). **(d)** The artery of Adamkiewicz (*arrows*) is identified by its straight course and rostral direction immediately after penetrating the dura just deep to the dural penetration of the left seventh thoracic nerve root (*arrowheads*). The forceps retract the denticulate ligament (*white*) medially. **(e)** Postoperatively the dural fistula no longer opacifies. The arrow designates the abrupt termination of the vessel previously supplying the AVF, and the arrowheads indicate the patent artery of Adamkiewicz and anterior spinal artery. (Reproduced with permission from Oldfield EH and Doppman JL, Spinal arteriovenous malformations. Clin Neurosurg 1988;34:161–183.)

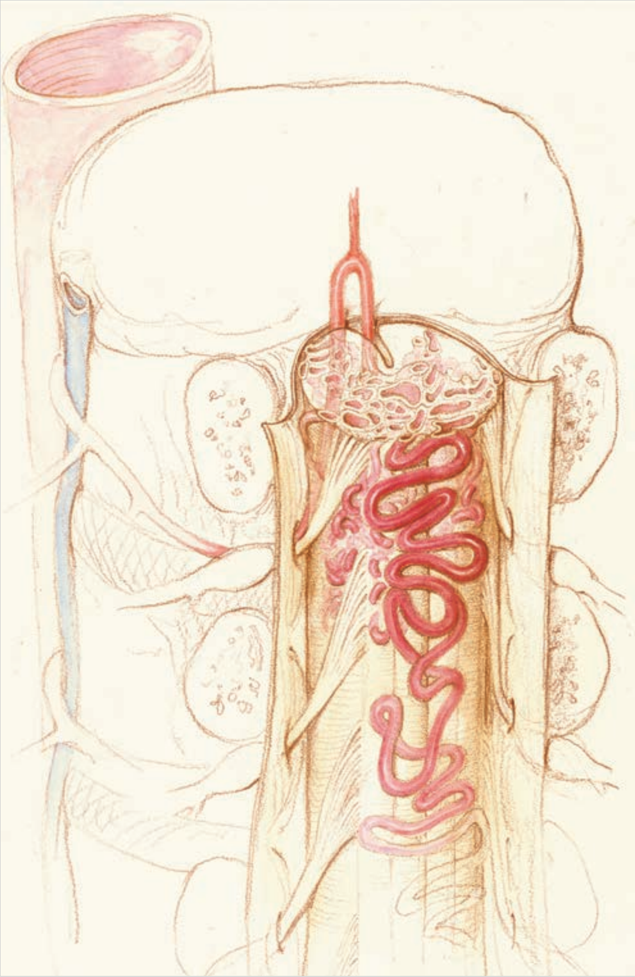

Fig. 30.4 Vascular anatomy of a juvenile intramedullary arteriovenous malformation (AVM) of the spinal cord. The juvenile type of intramedullary AVM is fed by enlarged medullary arteries via dilated anterior and posterior spinal arteries. The nidus of the AVM is extensive, often filling the spinal canal, and contains neural tissue within the interstices of the vessels of the AVM.

30.3.4 Cavernous Malformations

Cavernous malformations of the spine are usually diagnosed after they cause progressive, stuttering myelopathy by a cycle of repeated small intramedullary hemorrhages (▶ Fig. 30.8). Symptomatic lesions typically demonstrate recent hemorrhage surrounded by a hemosiderin-stained gliotic capsule at surgery and/or a hemosiderin ring on magnetic resonance imaging (MRI, ▶ Fig. 30.9). Recent information indicates that the natural history of spinal cavernous malformations is not as severe as was once thought, because patients with symptomatic spinal cavernous malformations, who are treated nonoperatively, have a small risk of clinically significant recurrent hemorrhage. When surgery is necessary in a patient with progressive neurological deterioration, the best approach to the lesion is via a route that transgresses the least amount of spinal cord tissue.

The decision how best to manage these lesions requires the usual considerations for surgery of intraparenchymal mass lesions of the spinal cord. In comparison to ventrally located lesions, cavernous malformations lying in the dorsal half of the cord are immediately accessible with a limited, linear myelotomy over the most superficial aspect of the lesion, require less manipulation of the spinal cord for exposure, and are associated with less risk of incurring additional neurological injury during surgery and a more favorable prognosis for improvement after removal.

30.4 Postoperative Management Including Possible Complications

Patients are monitored postoperatively for up to 24 hours in an intensive care unit. Longer stays may be necessary for some patients. The use of perioperative steroids is at the discretion of the surgeon. There is no compelling evidence of its benefit. Postoperative arteriography is typically performed shortly after surgery. Acute neurological deficits after surgery are investigated immediately with MRI, which defines potentially remediable postoperative problems such as compressive extradural or intraspinal hematoma and selective spinal arteriography. Hematomas should be evacuated if they cause spinal cord compression with neurological deficit. Residual malformation on postoperative angiography may require repeat surgery unless the goal of surgery was partial treatment and this was accomplished.

The outcome after treatment of dural AVFs, intradural spinal AVMs, and cavernous malformations is dependent not only on the type and location of the lesion but also on the preoperative neurological function. Patients who are ambulatory before treatment are usually ambulatory after treatment.

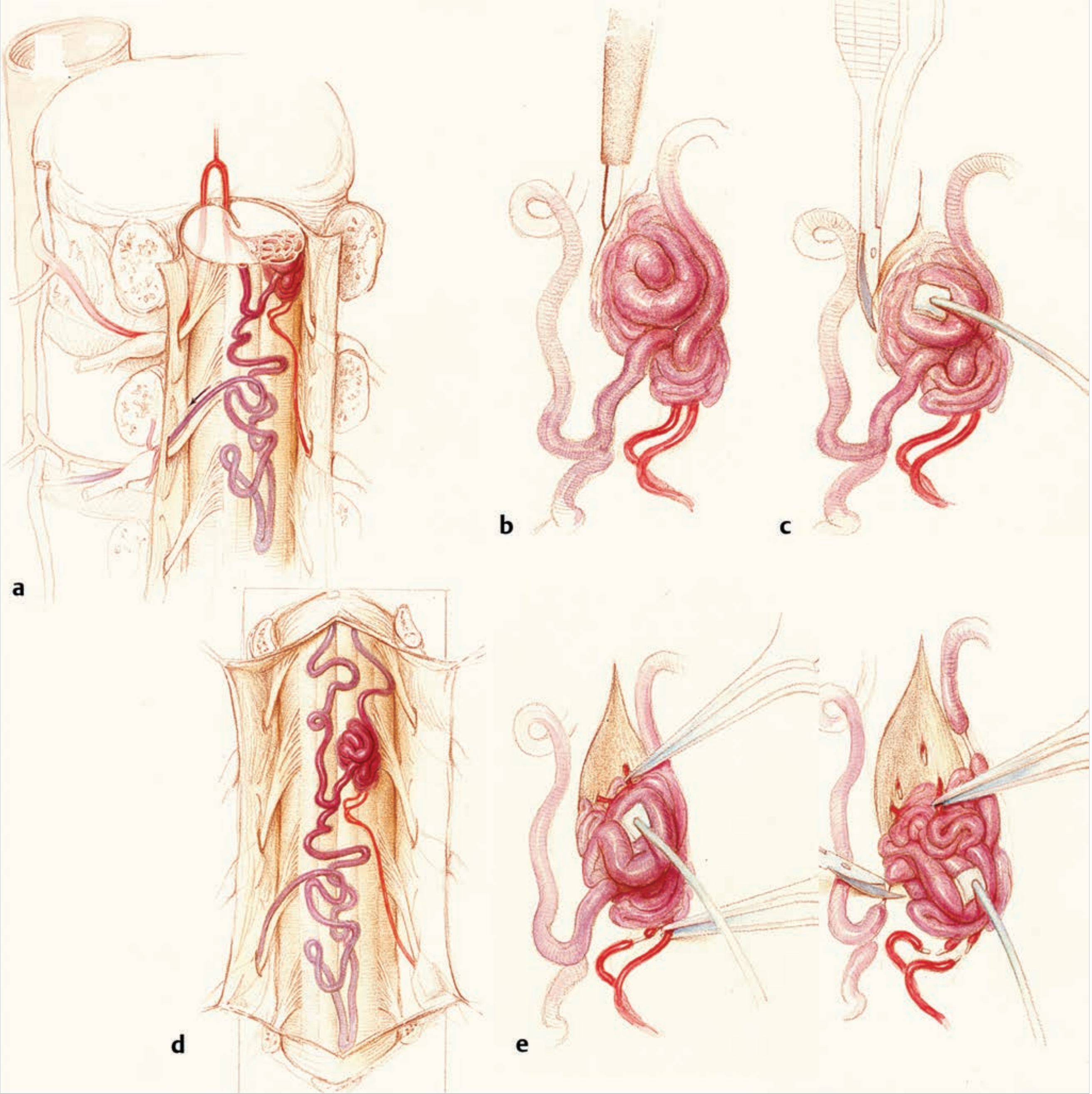

Fig. 30.5 **(a)** Vascular anatomy of glomus intramedullary arteriovenous malformation (AVM). The nidus of the glomus type of intramedullary AVM is a tightly packed nidus of blood vessels confined to a limited segment of the spinal cord. **(b)** After separating the arachnoid from the AVM, a diamond knife is used to incise the pia at the edge of the nidus. **(c)** To obtain the necessary exposure the pial incision is extended a few millimeters rostrally and caudally. **(d)** The superficial feeding vessels are interrupted sharply after they have been coagulated. **(e)** Resection of the AVM using a standard microsurgical technique.

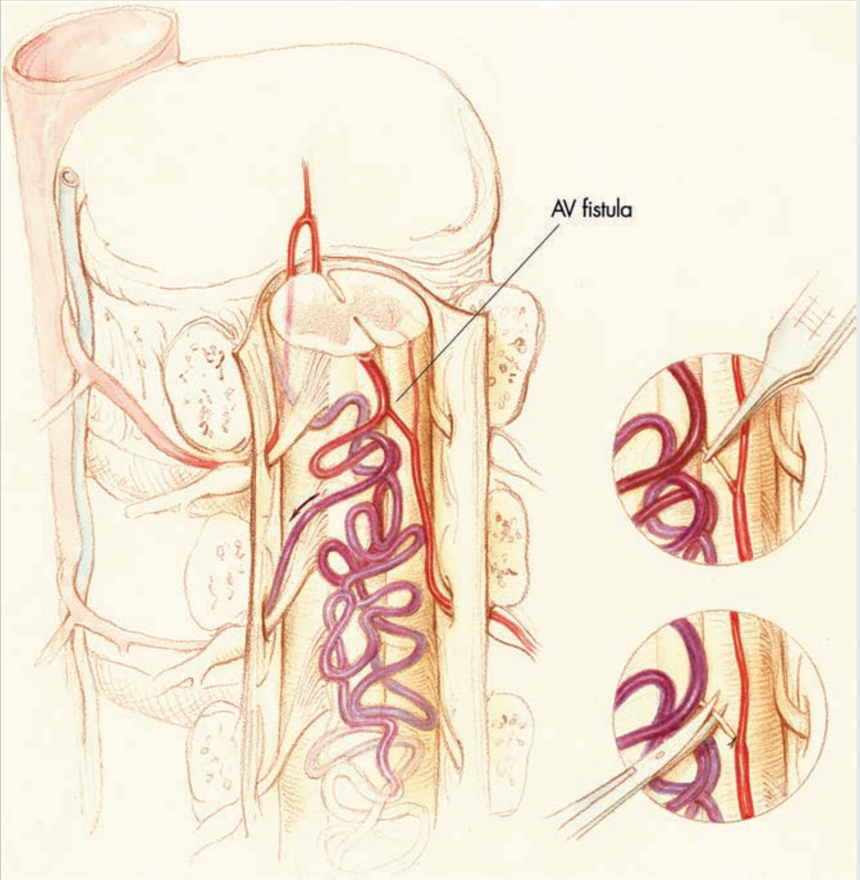

Fig. 30.6 Vascular anatomy of an intradural or perimedullary arteriovenous fistula (AVF). Medullary arteries provide the arterial supply, in this instance via a posterior spinal artery. Intradural AVFs often have associated arterial or venous aneurysms at the junction of the arterial and venous elements. Note the dilatation of the vein just distal to the AV shunt. The site of arterial to venous transition is identified and, after bipolar coagulation of a 4 to 6 mm segment of the distal portion of the artery(s) to the AVF, the AVF is interrupted on the distal portion of the arterial side of the fistula, beyond the last arterial branch to the spinal cord. In some instances a small clip or ligature is required, but most feeders can be managed by bipolar coagulation and sharp interruption alone. If an aneurysm or varix is the site of convergence of the feeding vessels, it is excised. Many of these lesions are not as simple as that illustrated here because these lesions can have more than one simple fistula in the same region of the pia, and the tortuosity and dilatation of the venous drainage often obscure the site of the AVF beneath a nest of blood vessels.

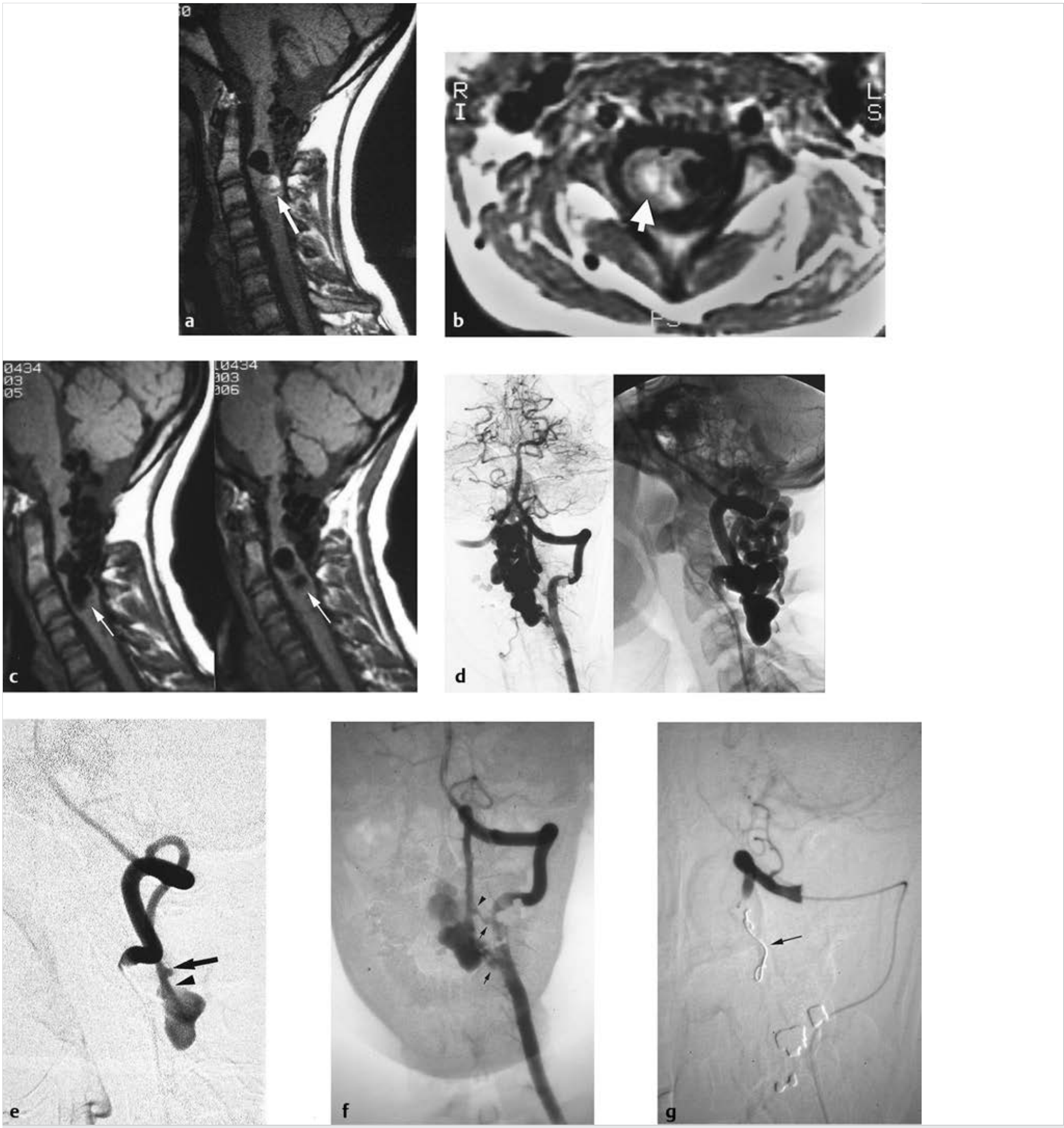

Fig. 30.7 An intradural arteriovenous fistula (AVF). **(a–c)** T1-weighted sagittal and axial magnetic resonance imaging (MRI) without contrast and **(d)** selective left vertebral arteriography using standard “cut films” (anteroposterior-left and lateral-right views) and **(e)** rapid sequence (10 frames/s) digital imaging (lateral view) of an AVF at the upper cervical segments of the spinal cord in a 17-year-old female with four hemorrhages but normal neurological function. An MRI scan 11 days after acute neurological changes **(a, b)** shows the patent (areas of low intramedullary and extramedullar signal) and thrombosed (high intramedullary signal, *arrows*) portions on an intramedullary varix and abnormal vessels in the upper cervical subarachnoid space and cisterna magna. Symptoms were attributed to thrombosis of one of the intramedullary varices **(a)**, thrombosis which was not present on an MRI scan performed 4 months previously **(c)**. Arteriography **(d)** suggested an arteriovenous malformation of the spinal cord with extensive blood flow, but rapid sequence (10 frames/s) imaging in **(e)** demonstrates that this is a simpler AVF (*arrowhead*) with an aneurysm (*arrow*) proximal to the AVF and a varix distal to the fistula. The varix was visible on the sagittal MRI on the left side of the spinal cord **(c**, left**)**. **(e, f)** Multiple feeding vessels converged at the site of the fistula, which was at the upper margin of the large intramedullary varix. In **(d)** the coiled, dilated arterialized veins conceal the fistula and obscure the aneurysm. **(g)** To reduce blood flow through the fistula, a coil was positioned in the distal portion of the principle feeding artery, a posterior spinal artery, just proximal to the aneurysm, the day before the fistula was interrupted surgically and the large varix was excised. Although the coil occluded the major flow to the AVF, small medullary arteries at the level of the second and third cervical vertebrae on the left **(f**, *arrows***)**

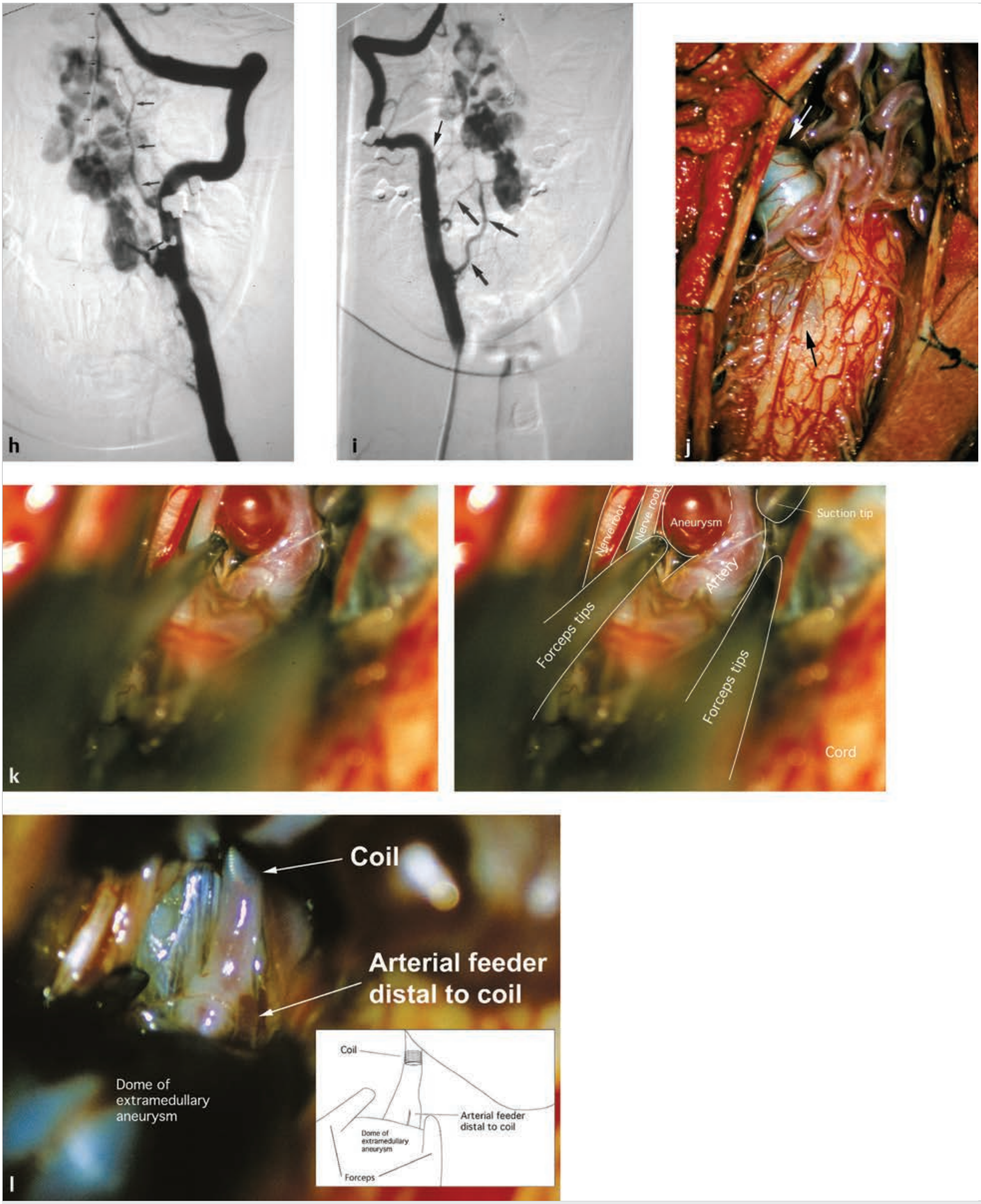
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Fig. 30.7 (*Continued*) and **(h**, *large arrows***)** and the second, third, and fourth on the right **(i**, *arrows***)** continued to supply the fistula by entering the major feeding vessel just distal to the coil. **(j)** At surgery, opening the dura and arachnoid exposed the extramedullar varix (*white arrow*), the blue–gray color of an intramedullary varix just beneath the pia (*black arrow*), and the tortuous arterialized veins overlying the spinal cord superiorly. **(k)** Exposure of the anterolateral margin of the spinal cord on the left side revealed the major feeding vessel and the small aneurysm that arose from it. **(l)** Dissection further rostrally exposed the bottom of the coil within the major feeding artery and an artery still feeding the AVF just distal to the coil. The medullary arteries still feeding the AVF were coagulated and interrupted. A vertical pial incision was made over the site of pial discoloration **(j**, *black arrow***)**. (*Continued*)

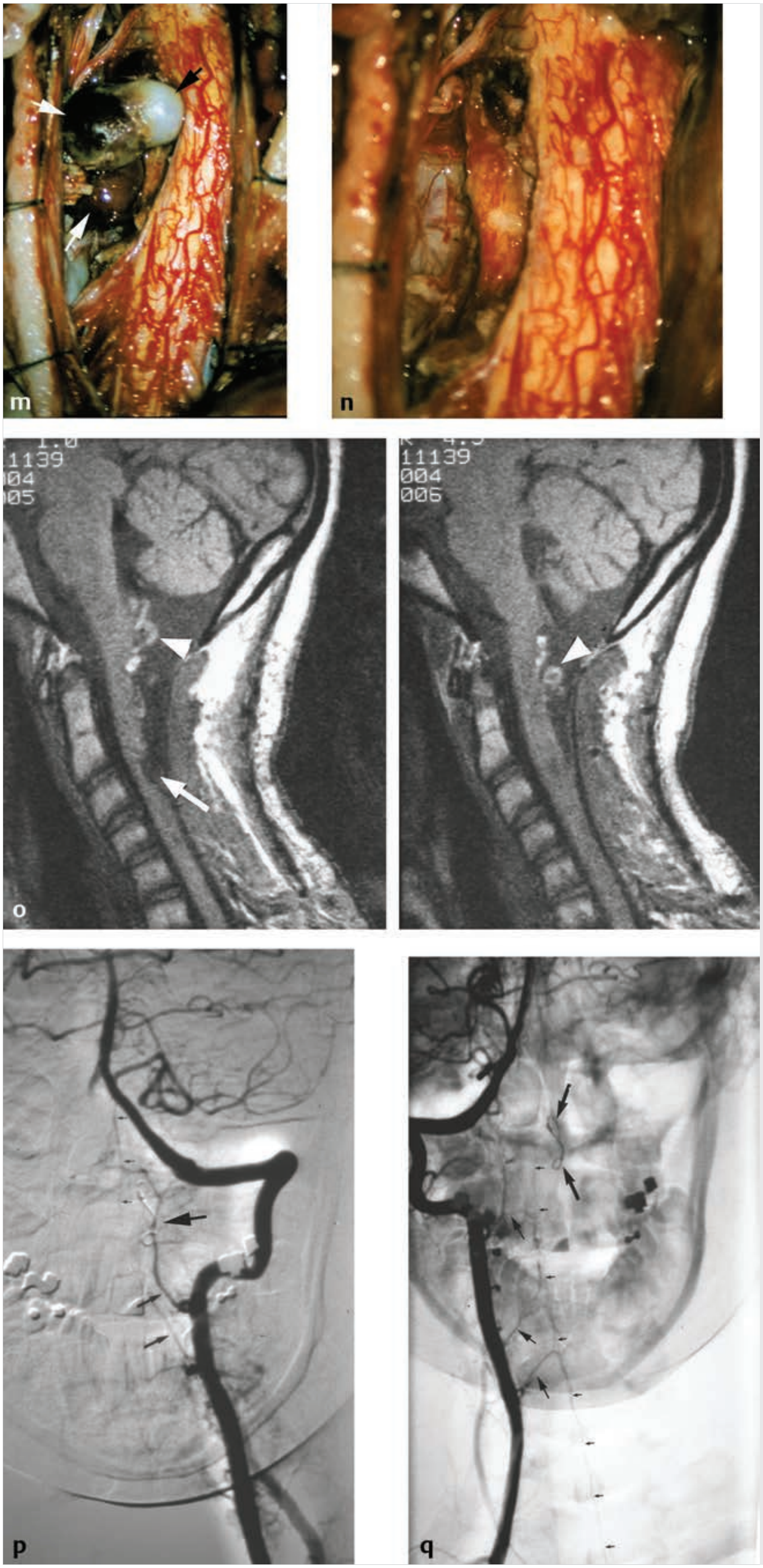

Fig. 30.7 (*Continued*) **(m)** The soft, gliotic tissue at the interface between the spinal cord and the aneurysm was used to dissect the aneurysm from the spinal cord, fully exposing the darkly colored patent *(white arrows)* and the gray–white thrombosed (*black arrow*) intramedullary portions of the aneurysm, and **(n)** the fistula was interrupted and the aneurysm excised. The dissection required rotation of the aneurysm such that in **(m)** the extramedullar component of it **(j**, *white arrow***)** is ventral and cannot be seen. **(o)** After surgery sagittal T1-weighted MRI revealed no abnormal signal void at the site of the previous aneurysms (*arrow*), and thrombosis of some of the abnormal veins (*arrowheads*). **(p)** Arteriography of the left and **(q)** right vertebral arteries confirmed patency of the anterior spinal artery. The large arrows in **(p)** and **(q)** indicate the coil, the intermediate-sized arrows show the ventral medullary arteries supplying the anterior spinal artery.

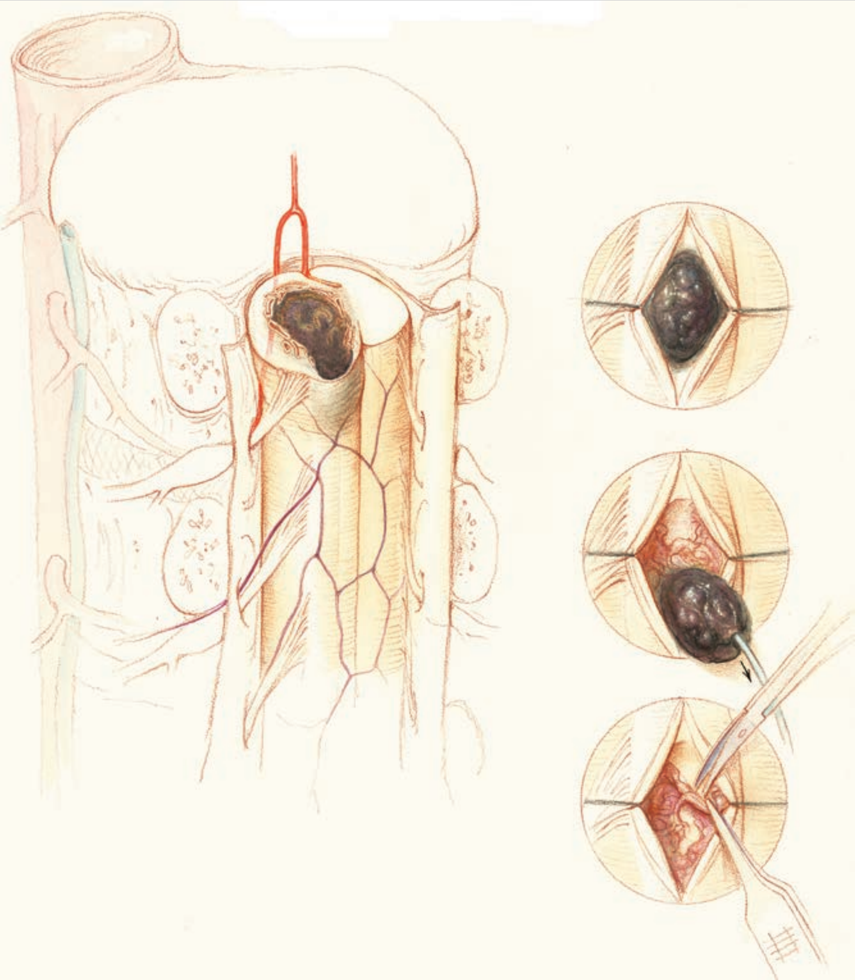

Fig. 30.8 Cavernous malformation of the spinal cord being easily resected using standard microsurgical technique.

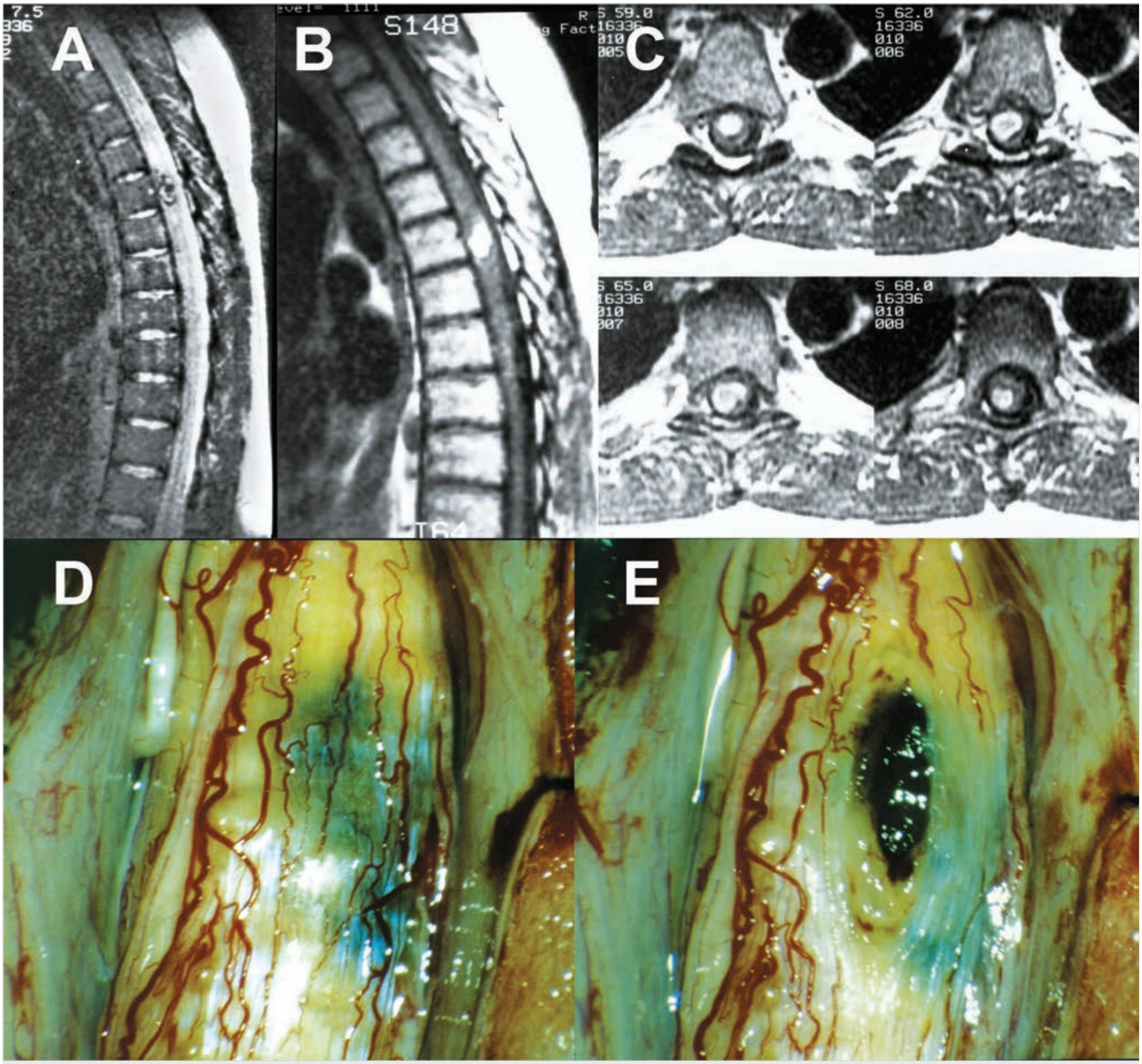

Fig. 30.9 Magnetic resonance imaging (MRI) and surgical findings of cavernous malformation of the thoracic spinal cord. **(a)** T2-weighted sagittal **(b)** T1-weighted sagittal and **(c)** axial MRI scans demonstrate a well-delineated intramedullary lesion of mixed signal, predominantly low-signal surrounding scattered areas of increased signal on T2-weighted MRI **(a)**. The patient presented with chronic progressive paraparesis. The high signal on T1-weighted MRI **(b, c)** is consistent with **(d, e)** methemoglobin accumulation in the subacute intramedullary hematoma, which was confirmed at surgery. **(d)** After opening the dura and arachnoid the blue pial discoloration overlying the malformation and intramedullary hematoma are typical of these lesions. **(e)** A linear pial incision over the superficial aspect of the angioma and intramedullary hematoma permits access to them for excision with minimal manipulation of the spinal cord.

31 Carotid Cavernous Fistulas

Joshua W. Osbun, C. Michael Cawley, Jacques E. Dion, and Daniel L. Barrow

Abstract

Carotid cavernous fistulas (CCFs) are abnormal arteriovenous shunts between the cavernous internal carotid artery (ICA) and its intracavernous branches and/or external carotid artery (ECA) and the venous channels of the cavernous sinus. These fistulas become symptomatic by causing venous hypertension in the cavernous sinus. Therefore, the venous drainage pattern determines the clinical nature of the condition. Significant morbidity can be associated with this uncommon vascular disorder because of the proximity of the cavernous sinus to many neurovascular structures (portions of cranial nerves III, IV, V [V1 and V2], and VI, the sympathetic plexus, a segment of the ICA and its intracavernous branches, subarachnoid space, and interconnectivity to a plexus of cerebral veins, some even distant from the sinus). This chapter reviews the specific types of treatments necessary for the different types of CCFs.

Keywords: carotid cavernous fistula, dural arteriovenous fistula, intracranial vascular malformation

31.1 Introduction

Carotid cavernous fistulas (CCFs) are abnormal arteriovenous shunts between the internal carotid artery (ICA) and the cavernous sinus either via direct connection with the ICA and/or its intracavernous branches or via external carotid artery (ECA) branches and the venous channels associated with the cavernous sinus. In keeping with the pathophysiology of most intracranial arteriovenous fistulas, CCFs as well cause symptoms via venous hypertension and exert their effect on surrounding tissue by venous engorgement and mass effect on adjacent structures. The venous drainage pattern therefore becomes an important anatomical feature that determines symptomatology. As such, CCFs can manifest themselves as proptosis and injection of the globe resulting in vision loss from increased intraocular pressure or by compression on cranial nerves passing through the cavernous sinus. Additionally, some may exhibit leptomeningeal retrograde venous drainage via collateral venous drainage between the cavernous sinus and sylvian veins, creating risk for intracranial hemorrhage.

31.2 Patient Selection

31.2.1 Classification

A useful classification for CCFs emphasizes the venous drainage pattern of fistulas and correlates with the clinical behavior observed in natural history studies.[1,2] The Borden classification of dural arteriovenous fistulas is only partly applicable to CCFs. In this system, type 1 lesions have drainage only into a dural sinus or meningeal veins, type 2 fistulas drain into a dural sinus and leptomeningeal veins, and type 3 lesions drain into leptomeningeal veins only. Those fistulas with leptomeningeal venous drainage (Borden types 2 and 3) are associated with an aggressive natural history and high incidence of hemorrhage or progressive neurological complications. The risk is high—nearly 20% annually with mortality of about 10% for such fistulas that have a history of hemorrhage. Emerging data suggest that even the Borden 2 and 3 fistulas may have a relatively benign natural history if they are diagnosed in patients with no evidence of a prior hemorrhage.[3]

In general, fistulas draining exclusively into a dural sinus (Borden type 1) have a benign natural history. CCFs are one exception to this generalization and they have unique characteristics and are better classified by other systems (▶ Fig. 31.1, ▶ Fig. 31.2, ▶ Table 31.1).[1] By inducing cavernous sinus hypertension, CCFs may lead to orbital venous engorgement that can cause pain, pulsatile exophthalmos, exposure keratopathy, conjunctival chemosis, and ocular motility restriction. These symptoms may progress to retinal ischemia/detachment, glaucoma, and ultimately visual loss.

31.2.2 Indications for Treatment

Treatment decisions are based on the local orbital symptomatology and by the pattern of venous drainage. Noninvasive treatment options such as observation and ipsilateral carotid compression may be appropriate for Borden type 1 lesions without objective ocular impairment or raised intraocular pressure (usually Barrow types B to D). Once leptomeningeal venous drainage, ocular impairment due to venous hypertension (chemosis, exophthalmos, ocular motility restriction, etc.) or orbital venous hypertension is detected, treatment is indicated to prevent hemorrhagic complications or visual loss.

The timing of treatment depends on the presenting features. Urgent treatment is indicated for CCFs with rapidly progressing visual compromise, markedly increased intraocular pressure (> 40 mm Hg) or focal neurological deficits other than cavernous sinus symptoms (usually secondary to leptomeningeal venous hypertension). Because of the high risk of hemorrhage from Borden 2 and 3 lesions, timely treatment of these is also indicated. A rare emergent complication is epistaxis, which can be torrential if there is a fracture or erosion through the thin sphenoid sinus wall.

31.2.3 Treatment Options

The goal of treatment of most fistulas, including CCFs ideally should be to obliterate the arteriovenous shunt at the site of fistula while preserving patency of the cerebral arteries, in this case the ICA. Treatment options include observation, carotid compression, transarterial and/or transvenous embolization, and microsurgical approaches to facilitate embolization or accomplish venous disconnection. Because of the benign course of those CCFs draining exclusively into the cavernous sinus (Borden type 1), such patients can be managed with observation or intermittent carotid compression. Manual carotid compression is done six times a day for 30 seconds each session over a period of 4 to 6 weeks. It is very helpful to demonstrate the technique to the patient with instructions to use the contralateral hand to compress the carotid artery in the neck. If this

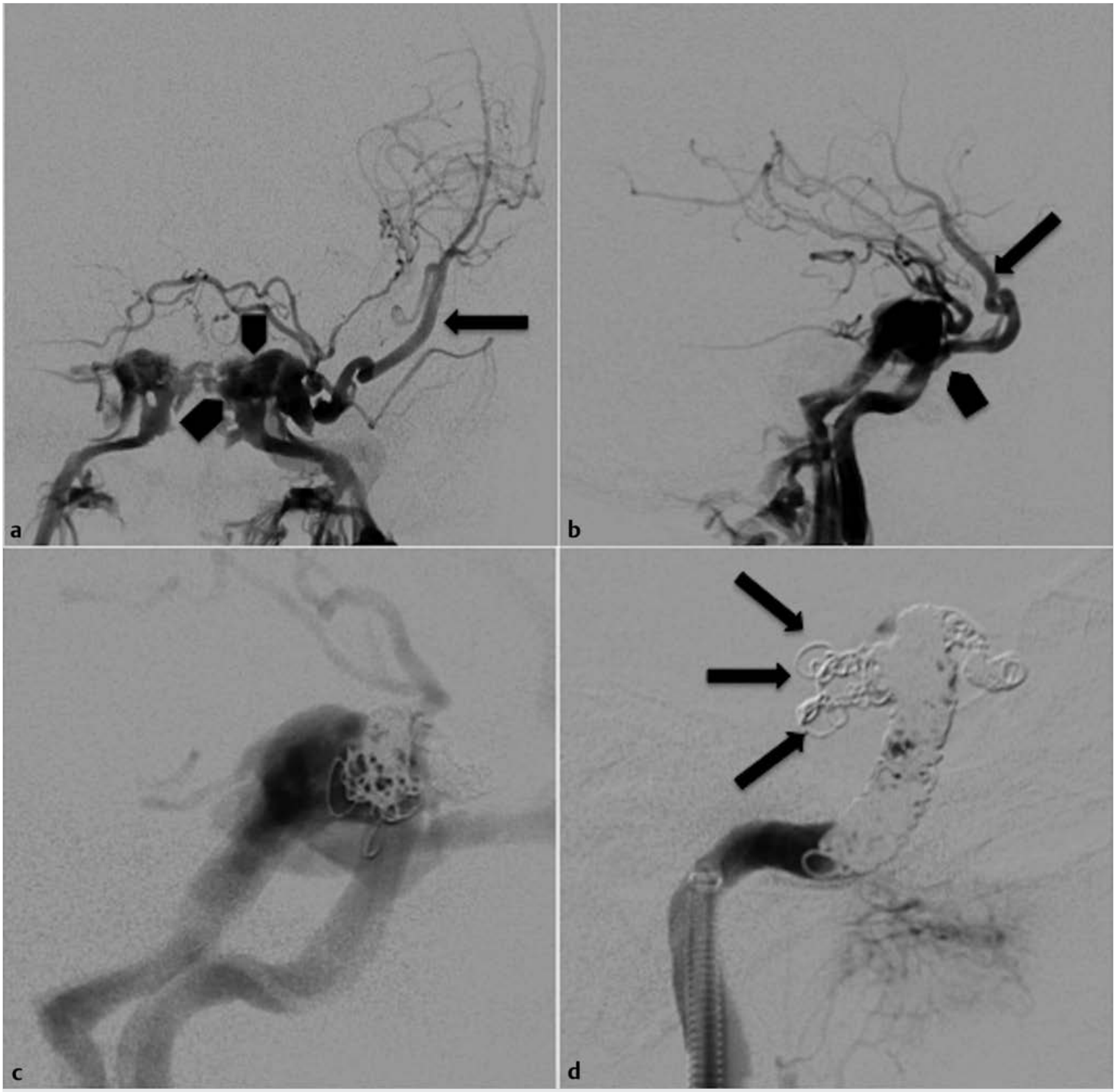

Fig. 31.1 A 66-year-old woman presented with progressive left eye redness, proptosis, and a bruit 1 month after a motor vehicle crash. Left internal carotid angiogram demonstrates a direct CCF (Barrow type A) with venous drainage mainly through the inferior petrosal sinus in the anteroposterior **(a)** and lateral **(b)** projections, with arrowheads demonstrating the point of fistula and arrows demonstrating an additional cortical draining vein. **(c)** The fistula was treated endovascularly with transarterial coil embolization of the distal ICA and coil occlusion of the defect. **(d)** Final lateral angiogram demonstrating obliteration of the fistula, with arrows demarcating the "hole" in the carotid artery where coils herniate into the cavernous sinus.

causes cerebral ischemia, the compressing extremity weakens and the manual pressure stops. The patient should be sitting to prevent a fall resulting from a vasovagal attack. Stop antiplatelet or anticoagulation treatment during this period. To induce thrombosis, the carotid and jugular flows should be compressed together and any bruit should disappear. Close neurological and neuro-ophthalmological follow-up is important. This includes visual acuity, pupillomotor activity, intraocular pressure, visual fields, proptosis measurement, gonioscopy, and direct and indirect fundoscopy. Occasionally spontaneous resolution of benign symptoms, like bruit, is caused by conversion of venous drainage solely into the cavernous sinus to a leptomeningeal route, signifying the transformation of a benign dural arteriovenous malformations (DAVMs) to an aggressive type. On the other hand, resolution of symptoms can also mean resolution of the fistula, which can occur spontaneously or after diagnostic angiography alone.

For patients with neurological or serious ophthalmic dysfunction, or when they harbor Borden type 2 or 3 lesions, aggressive therapy is indicated. We recommend treatment for

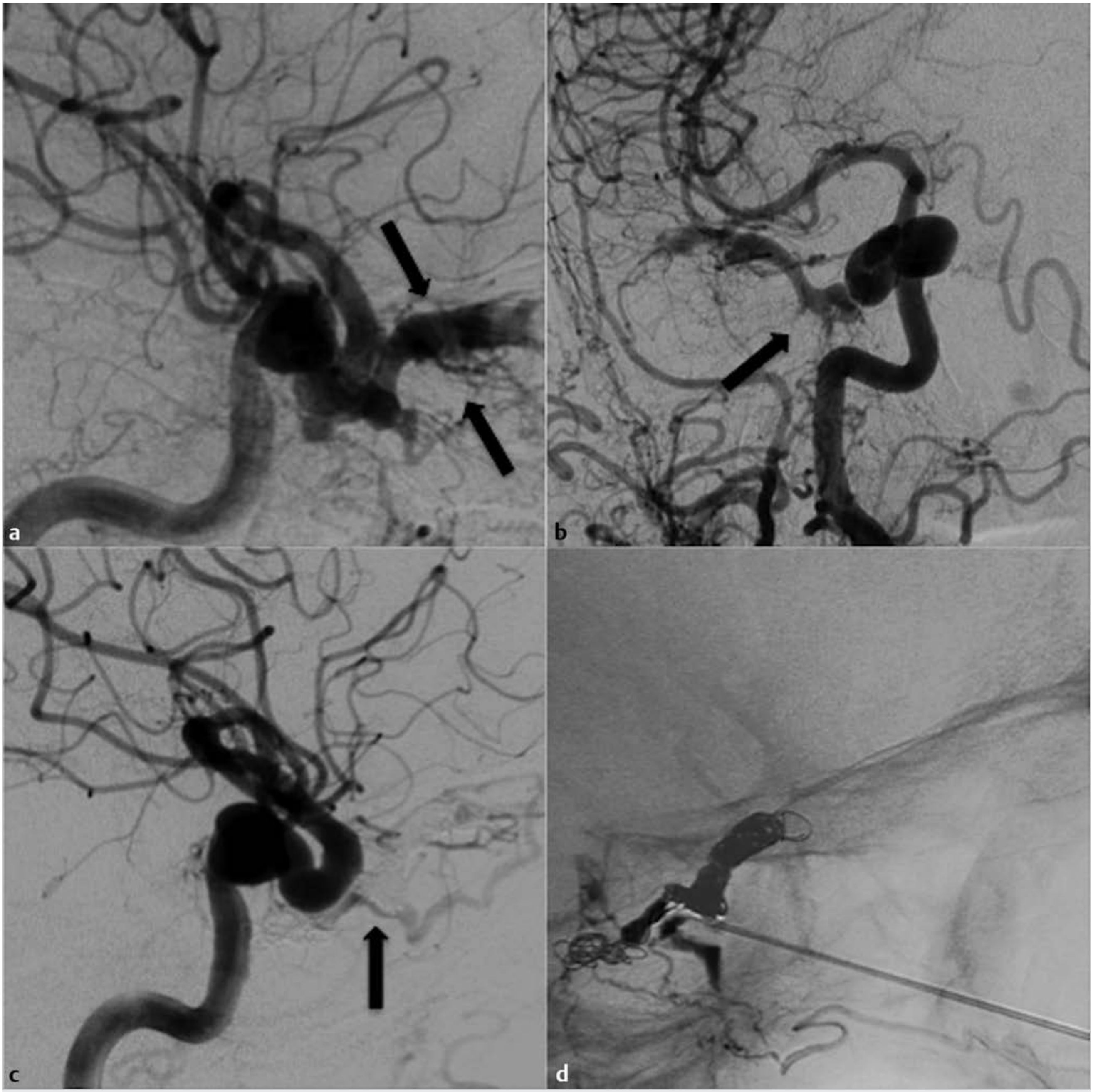

Fig. 31.2 An 80-year-old woman presented with progressive right eye redness and exophthalmos. **(a)** and **(b)** Cerebral angiogram demonstrates an indirect CCF fed by cavernous branches of the ICA in addition to ECA branches (Barrow type D). Arrows demarcate the SOV with small ECA feeders. Initially this patient was treated with transvenous coil embolization of the SOV and cavernous sinus. **(c)** Follow-up angiogram demonstrated a persistent fistula with draining vein (arrow) and pathways for further attempts at transvenous embolization were blocked by the previous treatment. The patient underwent direct orbital puncture for access to the cavernous sinus. **(d)** The fistulous pouch was embolized with ethylene vinyl alcohol copolymer. (Onyx, Micro Therapeutics, CA)

visual deterioration, diplopia related to vascular engorgement and enlargement of the extraocular musculature or to neural compression within the cavernous sinus, intolerable bruit or headache, and/or severe proptosis with refractory exposure keratopathy. Transvenous embolization is the preferred treatment, although transarterial embolization is a reasonable alternative if all fistulous feeders are from the external carotid system.

Transarterial embolization was previously a major treatment for DAVMs. The cure rates range from 70 to 80%. At present, this option is chosen for lesions with predominant ECA feeders and in which venous embolization is not feasible due to either poor access or high risks. Transarterial closure of direct CCFs with detachable coils with or without the addition of liquid embolic agents is also well described.[4,5,6,7] In patients who tolerate balloon test occlusion on the side of the fistula, carotid sacrifice with coil placement both proximal and distal to the site of the fistula can be very effective. Combination of transarterial embolization with stereotactic radiosurgery has been reported.

The transvenous approach has become the treatment of choice for most DAVMs, including CCFs. Difficulty may be encountered from multiple peripheral valves when catheterizing the femoral vein. From the internal jugular vein, the inferior petrosal sinus is the most common access to the cavernous sinus. Occasionally, microguidewires and microcatheters can be maneuvered through a thrombosed inferior petrosal sinus. Many other access routes have been described, including the superior ophthalmic and angular veins, superior petrosal sinus, pterygoid plexus, and dilated cortical draining veins. Direct percutaneous puncture of the SOV and even the cavernous sinus through the superior orbital fissure has also been performed. Detachable coils are typically used for embolization. Complete packing of the cavernous sinus can be tolerated, although some have been successful at preserving the normal sylvian drainage into the sinus while obliterating the fistula.

Table 31.1 Classification of carotid cavernous fistulas according to Barrow et al[1]

Category	Characteristics
A	Direct fistula between internal carotid artery and cavernous sinus, usually secondary to trauma or occasionally to rupture of a cavernous carotid aneurysm (▶ Fig. 31.1).
B	Shunts between meningeal branches of the cavernous internal carotid artery, namely the meningohypophyseal trunk, capsular arteries and inferior lateral trunk (▶ Fig. 31.2).
C	Shunts between the dural branches of the external carotid artery and the cavernous sinus (▶ Fig. 31.2).
D	Complex lesions involving shunting from meningeal branches of both the cavernous ICA and ECA (▶ Fig. 31.2).

While transvenous and transarterial access to the cavernous sinus through standard endovascular techniques has yielded excellent results and is probably the first treatment option, on occasion conventional endovascular routes may not be available either as a result of venous stenosis or occlusion, or from surgical carotid occlusion or fistula trapping. In some instances an engorged superior ophthalmic vein (SOV) may be present, allowing direct surgical exposure and access to the cavernous sinus. In the challenging circumstance of a completely trapped CCF without ophthalmic venous outflow, the cavernous sinus can be accessed surgically through a modified pterional craniotomy with pretemporal extradural dissection. A less invasive approach is a percutaneous transorbital, infraocular cavernous sinus puncture via the superior orbital fissure (transorbital puncture).

31.3 Preoperative Preparation

Thorough angiographic investigation is a prerequisite for treatment. Lateral series are the most valuable, although the anteroposterior series of the ipsilateral ICA is performed to show any reflux into cortical veins and across the circular sinus to the opposite side. Contralateral carotid angiograms may help to detect bilateral CCFs, significant stenosis, occlusion, or aneurysm that may complicate possible ipsilateral carotid sacrifice. An ipsilateral vertebral artery injection can establish the level of the fistula, as well as the collateral circulation of the circle of Willis.

31.4 Operative Procedure

31.4.1 Surgical Exposure of the Superior Ophthalmic Vein

A 15-mm incision is made at the medial lid crease or the subbrow region after infiltration of a local anesthetic with epinephrine. Blunt dissection is used to identify the orbital septum (▶ Fig. 31.3). Just beneath the superior orbital rim at the level of

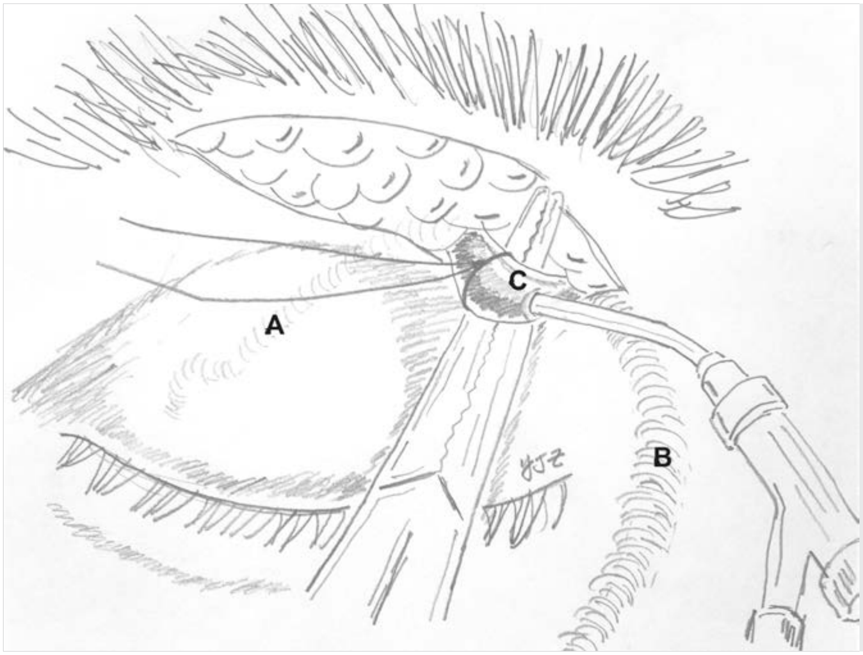

Fig. 31.3 Surgical superior ophthalmic vein exposure. Upper figure: The lid-crease incision followed by orbital septum division provides access to the superomedial orbit. A, lid-crease incision; B, orbicularis muscle; C, orbital septum; D, orbital fat. Lower figure: The superior ophthalmic vein is lateral to the trochlea. The vein has been isolated by blunt dissection and cannulated for embolizing the cavernous sinus. A, superior ophthalmic vein leading to the cavernous sinus; B, angular branch of the facial vein; C, exposed segment of the superior ophthalmic vein, which has been canalized by an angiographic catheter.

the trochlea, the orbital septum is incised along the width of the skin incision, with the trochlea and superior oblique tendon identified and protected. The orbital fat is retracted laterally with blunt dissection. In the medial orbit just posterolateral to the trochlea the SOV will appear (▶ Fig. 31.3). Frequently, the vein is arterialized and quite fragile. The vein is skeletonized with blunt dissection, and two 2–0 silk ligatures are placed around the vessel for manipulation and retraction. Between the ligatures, venipuncture is performed and a mandrel guidewire (Cook, Bloomington, IN) is introduced, followed by a 4 French micropuncture introducer (Gait Medical Corp., Garland, TX) for SOV catheterization. An additional suture through the periosteum of the arcus marginalis can be used to secure the cannula. A standard 4 French diagnostic catheter is linked to the introducer (▶ Fig. 31.3), so that embolization through a microcatheter can be performed with standard endovascular techniques with better ergonomic control and less radiation exposure to the clinicians.

Upon completion of the embolization procedure, the silk ligature is loosened and the catheter is removed. At this point the vein should no longer be arterialized, so there is minimal flow. The vessel is coagulated and divided. The deep tissues and orbital septum are left open and repositioned. The skin incision is closed with a running 6–0 chromic or polypropylene suture, which is typically removed on postoperative day 5.

31.4.2 Transcranial Surgical Exposure of the Superior Orbital Fissure

The pretemporal extradural region is exposed through a modified pterional approach that incorporates more medial subfrontal extension. After elevating the pterional bone flap, the lateral sphenoid wing is drilled along its medial extension. The meningo-orbital artery is coagulated and divided. This allows further separation of the dura of the temporal fossa floor from the dura overlying the region of the cavernous sinus and extending anteriorly to the superior orbital fissure. The exposure of this area is further enhanced by drilling the posterior aspect of the orbital roof all the way to the lateral aspect of the anterior clinoid process (▶ Fig. 31.4). A modified orbitozygomatic osteotomy or full orbitozygomatic osteotomy incorporating the maxilla and root of the zygoma may facilitate freedom of instrument movement and pretemporal/subtemporal exposure particularly around trigeminal nerve divisions. After exposing the superior orbital fissure region, microsurgical dissection is used to open the periorbita and to identify the cranial nerves. At this stage, a Doppler probe is used to localize the high arterial flow of the fistulized ophthalmic veins at the anterior aspect of the cavernous sinus. Indocyanine green angiography can also aid in characterization and localization of the fistula. Guided by these adjuncts, the high-flow venous channel is cannulated and a mandrel guidewire (Cook, Bloomington, IN), threading posteriorly toward the cavernous sinus (▶ Fig. 31.4). Over the guidewire, a 4 French micropuncture introducer (Galt Medical Corp., Garland, TX) is placed, and a microcatheter is then introduced into the cavernous sinus to initiate embolization for fistula occlusion. Once intraoperative arteriography confirms complete obliteration of the fistula, the catheters are removed and bleeding from the cavernous sinus puncture can be controlled with Gelfoam packing and gentle pressure with a cottonoid as the cavernous sinus should no longer be arterialized and will return to a low pressure venous system. The craniotomy is then closed in the standard fashion.

31.4.3 Percutaneous Transorbital Intraocular Cavernous Sinus Puncture

A thorough understanding of the three-dimensional anatomy of the cavernous sinus is critical for this approach. The cavernous sinus is a large, parasellar venous sinusoid situated posterior to the orbital apex. Its lateral and superior

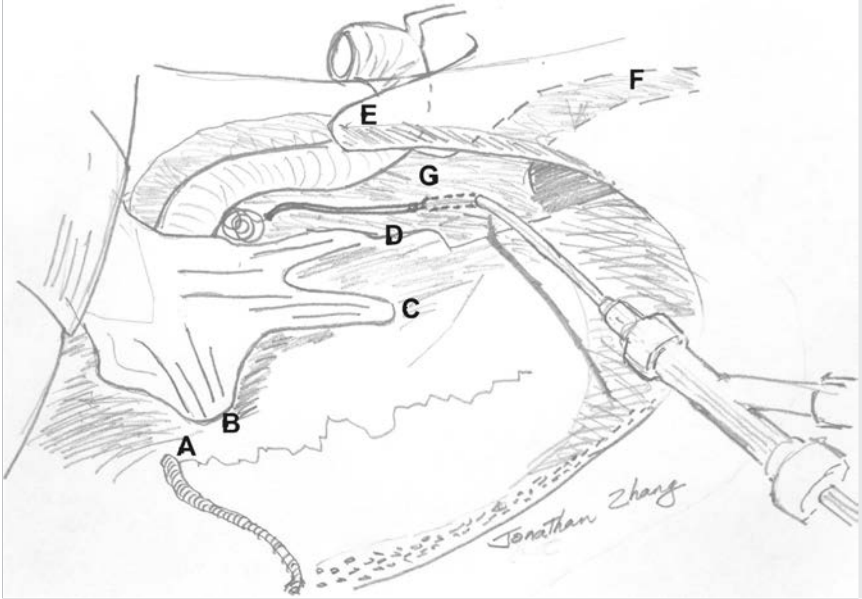

Fig. 31.4 Transcranial surgical exposure of the superior orbital fissure. Upper figure: Modified pterional craniotomy with posterolateral orbital roof removal, exposing the pretemporal region. A, partly removed lesser wing of the sphenoid ridge; B, anterior clinoid; C, periorbita, with partial orbital unroofing; D, dura overlying the superior orbital fissure and cavernous sinus; E, temporal fossa dura. Lower figure: Pretemporal extradural dissection exposes the lateral cavernous sinus and dura over the superior orbital fissure. A, foramen spinosum with divided middle meningeal artery; B, foramen ovale with V3 branch of trigeminal nerve; C, foramen rotundum with V2 division of trigeminal nerve; D, superior orbital fissure with V1; E, anterior clinoid process; F, superior ophthalmic vein, with retrograde venous congestion; G, anterior cavernous sinus, punctured and canalized by an angiographic catheter for embolization.

margins are bordered by dura mater, which separates it from the subarachnoid space. The superior boundary of the sinus extends posteriorly from the anterior clinoid process to the dorsum sellae. The inferior and medial borders are made up by the sphenoid bone. The ICA enters the cavernous sinus posteriorly and inferiorly as it crosses the foramen lacerum and exits medial to the anterior clinoid process, entering the subarachnoid space.

The superior orbital fissure is a slit-like opening between the lesser sphenoid wing superomedially and greater sphenoid wing inferolaterally (▶ Fig. 31.5**)**. The inferomedial portion of the superior orbital fissure is anterior to the cavernous sinus. Cranial nerves III, IV, and VI; the ophthalmic branch of cranial nerve V; and the lacrimal and orbital branches of the middle meningeal artery travel through the superior orbital fissure. Other than cranial nerve IV and the inferior division of the third

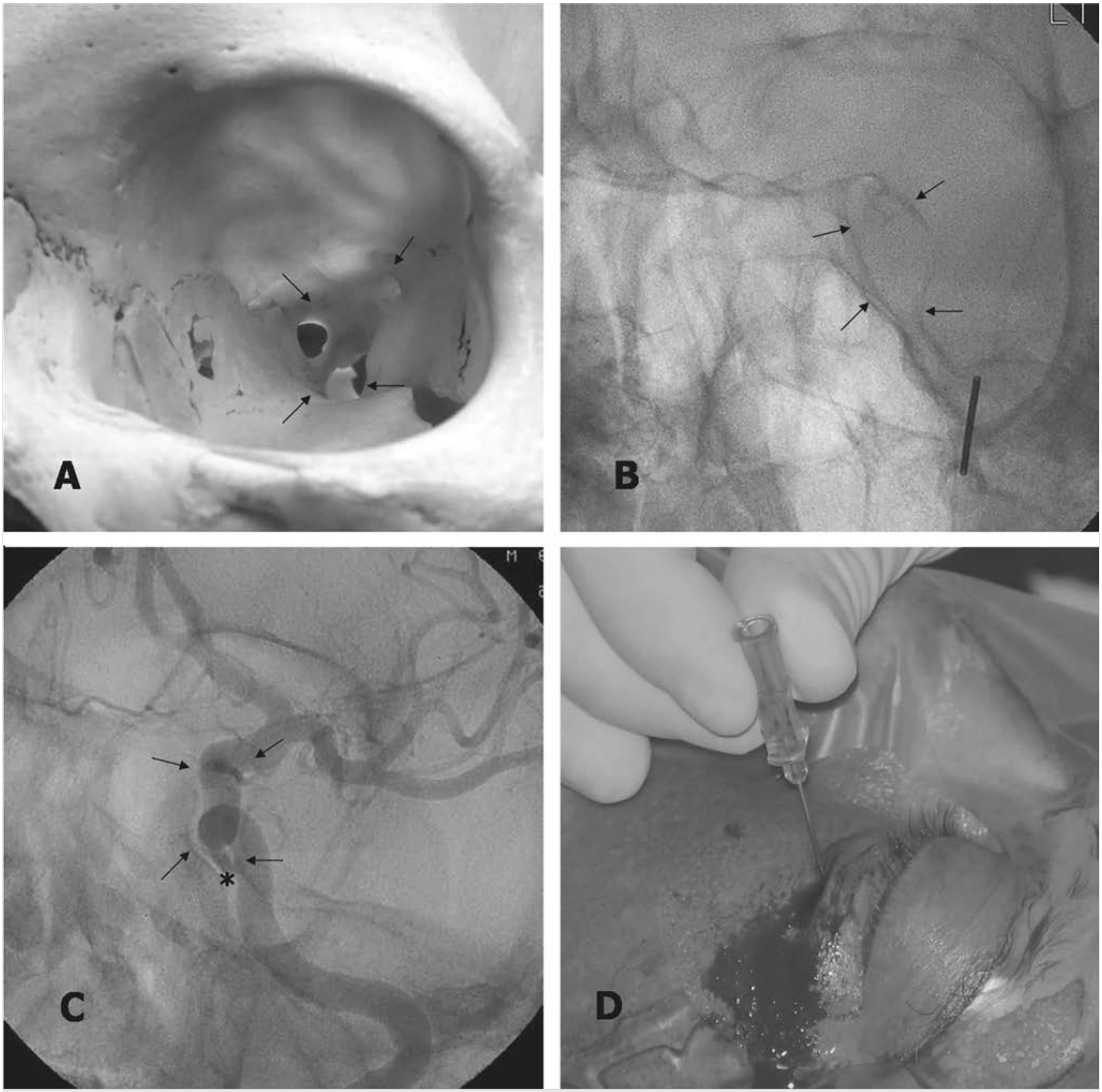

Fig. 31.5 Percutaneous, transorbital, intraocular, and transsuperior orbital fissure cavernous sinus puncture. **(a)** Skull specimen showing the left orbit with puncture trajectory and outline of the superior orbital fissure *(arrows)*. **(b)** Oblique fluoroscopic view of the left orbit with the superior orbital fissure highlighted (*arrows*); the puncture needle is advanced along the orbital floor, toward the inferolateral corner of the superior orbital fissure. **(c)** Same view of the left orbit with left internal carotid artery injection, revealing its relationship with the superior orbital fissure (asterisk represents the early venous drainage in the cavernous sinus caused by the fistula). **(d)** Intraocular puncture site and trajectory.

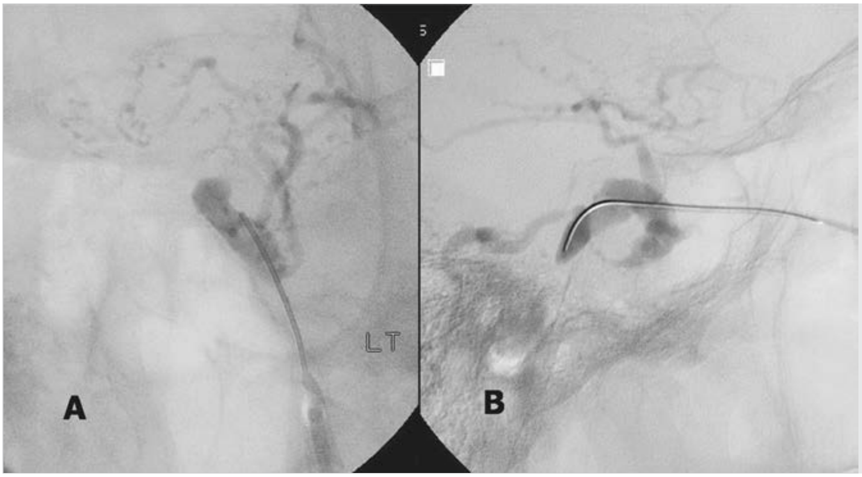

Fig. 31.6 **(a)** Frontal and **(b)** lateral projections of venogram upon percutaneous transorbital puncture and canalization of the cavernous sinus. Leptomeningeal venous drainage is shown.

cranial nerve, most of the superior orbital fissure contents are situated superolaterally. The ophthalmic artery and optic nerve pass through the optic canal, which is superior and medial to the superior orbital fissure.

The procedure is performed under general anesthesia with endotracheal intubation and high-resolution biplane fluoroscopic guidance. First the periorbital area is prepared and draped sterilely and a small skin incision is made along the inferolateral aspect of the ipsilateral eyelid. A 21 gauge, 7 cm puncture needle is advanced along the orbital wall posteriorly and inferiorly to avoid globe injury (▶ Fig. 31.5). Using fluoroscopic guidance and angiographic roadmap technique, the needle is directed along the inferolateral wall of the orbit, toward the superior orbital fissure. It is important to enter the cavernous sinus anteriorly to avoid subarachnoid space transgression. This can be facilitated by angling the fluoroscopic image intensifier in an oblique projection, oriented to the long axis of the orbit so that the extreme medial portion of the superior orbital fissure is visible and used as a target (▶ Fig. 31.5). Biplane angiography with roadmapping may be performed to opacify the cavernous sinus and confirm the target.

The needle is advanced through the superior orbital fissure into the cavernous sinus, and the correct position is confirmed with contrast injection (▶ Fig. 31.6). Using the Seldinger technique, the needle is then exchanged with a 4 French microintroducer (Galt Medical Corp., Garland, TX), which is in turn linked to a 4 French vertebral catheter (Cordis Corp., Miami, FL) through a Tuohy-Borst Y-adaptor (Cook Medical, Inc., Bloomington, IN). This system will allow ergonomic deployment of embolic agents and also increases the distance between the operators and the image intensifier to reduce radiation exposure. Standard endovascular coil embolization can then be employed to obliterate the cavernous sinus. Alternatively, liquid embolic agents including n-butyl cyanoacrylate (Trufill, Codman Neurovascular, NJ) and ethylene vinyl alcohol copolymer (Onyx, Micro Therapeutics, CA) can be injected through the access system. In some cases, particularly if a low flow fistula is present, the injection of D50 glucose solution is sufficient to thrombose and obliterate the lesion. Once control arteriography confirms complete obliteration of the fistula, the catheters can be removed and eyelid puncture site hemostasis achieved with gentle compression. Postoperative cranial neuropathy, orbital hematoma, vision loss, and subarachnoid hemorrhage are potential complications, although they are rare in experienced hands.

31.5 Postoperative Management Including Possible Complications

Patients who undergo craniotomy should be observed at least overnight in an intensive care setting. We do not routinely administer steroids or anticonvulsant medications. The main complications include cerebral ischemia due to occlusion of the ICA or its branches. This can occur if the cavernous sinus is packed too aggressively. The treatment depends on whether infarction has developed. Reestablishing flow might be indicated when there is ischemia but no infarction. Incomplete occlusion of the fistula and recurrence may occur and are managed by treatment if deemed necessary based on the indications discussed earlier. Posttreatment imaging should include a catheter angiogram to prove the lesion is obliterated, and subsequent imaging can be based on clinical findings. Cranial nerve palsies, most commonly of the III or IV nerves, may occur and usually resolve over several months. Overall, complication rates are generally reported to be 5%, including around 1% for cerebral infarction and less for diabetes insipidus, visual deterioration, and glaucoma. Rarely reported are choroidal detachment, blepharoptosis, and transient forehead dysesthesia. Operative mortality is rare.

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32 Transverse and Sigmoid Dural Arteriovenous Fistula

Johnny Wong, Rachel Tymianski, Vitor Mendes Pereira, Ivan Radovanovic, and Michael Tymianski

Abstract

Dural arteriovenous fistulae (DAVFs) are arteriovenous shunts located within the dural leaflets and constitute 10 to 15 % of intracranial vascular malformations. Transverse and sigmoid DAVFs are the most common locations for DAVF. Clinical presentations include pulsatile tinnitus, intracranial hemorrhage, and neurological deficits. Currently, both Borden and the Cognard classifications of DAVF have gained widespread popularity, and are based on patterns of venous drainage from the dural fistulae. Low-risk DAVFs (Borden I or Cognard I, IIa) have no retrograde leptomeningeal venous drainage and are associated with a relatively benign natural history.[1,2] In contrast, DAVF with retrograde leptomeningeal venous drainage (Borden II, III or Cognard IIb, III, IV) are associated with an increased risk of hemorrhage and neurological deficits. Treatment is therefore recommended for high risk DAVF, and its objective is to completely obliterate the arterialized draining vein. Available modalities include microsurgery, endovascular embolization (via transarterial or transvenous approaches), or radiosurgery. This chapter outlines the natural history of DAVF, and the indications and surgical techniques for treatment of transverse and sigmoid DAVF.

Keywords: dural arteriovenous fistula, transverse sinus, sigmoid sinus, microsurgery, leptomeningeal venous drainage

32.1 Introduction

Dural arteriovenous fistulae (DAVF) represent approximately 10 to 15% of all intracranial arteriovenous malformations.[3] The etiology of DAVF is not well understood, but associations with head trauma, dural sinus thrombosis, tumors, infections, or previous craniotomies have been identified.[2,4,5] Unlike brain arteriovenous malformations, DAVF are generally thought to be acquired lesions[5,6] and are associated with an annual hemorrhage risk of 1.5%.[6]

Common locations for DAVF include transverse-sigmoid sinus (40–50% of cases), cavernous sinus (16–20%), superior sagittal sinus (8–13%), tentorium (4–12%), and anterior skull base (4%).[1,6,7]

DAVF are characterized by dural arterial supply, either via single or multiple arterial feeders usually to a common fistulous point of drainage into a venous sinus or leptomeningeal veins.[8] In DAVF involving the transverse and sigmoid sinus, the arterial supply is mainly derived from branches of the external carotid artery, such as intraosseous branches of occipital artery, posterior auricular artery, middle meningeal artery, and ascending pharyngeal artery. Other sources of arterial supply include meningeal branches from the vertebral artery and the tentorial branch of the meningohypophyseal trunk (artery of Bernasconi–Cassinari) from the internal carotid artery. Venous drainage may be through ipsilateral transverse and sigmoid sinuses or through the contralateral side if the ipsilateral sinus is occluded. Cortical draining veins may be involved when arterialized blood flows in a retrograde direction through leptomeningeal veins, resulting in venous hypertension, congestion, and hemorrhage.[8]

32.2 Classification, Presentation, and Natural History

The most popular classifications of DAVF are those of Cognard et al,[1] and Borden et al (▶ Table 32.1).[2] The Cognard classification has five categories based on the following angiographic features: Involvement and direction of flow through venous sinuses, presence of retrograde leptomeningeal venous drainage (or cortical venous reflux), venous ectasia/pouches or drainage into spinal veins.[1] The Borden classification is a simpler system: type I has dural venous sinus drainage only (▶ Fig. 32.1**a**, ▶ Fig. 32.2**a**), type II contains both sinus and retrograde leptomeningeal venous drainage (▶ Fig. 32.1**b**, ▶ Fig. 32.3**a**), while in type III, drainage occurs through retrograde leptomeningeal venous drainage without sinus involvement (▶ Fig. 32.1**c**, ▶ Fig. 32.4**b**).[2]

Table 32.1 Radiological and clinical features of DAVF according to Borden and Cognard classifications

Classification		Radiological features		Clinical features
Borden[2]	Cognard[1]	Sinus Involvement	Cortical Venous Reflux	Clinical Presentation
I	I	Anterograde	No	Pulsatile tinnitus; 2% present as hemorrhage or neurological deficit Annual hemorrhage risk: 2%
	IIa	Retrograde		
II	IIb	Retrograde	Yes	39% present as hemorrhage or neurological deficit Annual hemorrhage risk: 8.1%
III	III	None	Yes	79% present as hemorrhage or neurological deficit Annual hemorrhage risk: 8.1%
	IV		Yes, with venous ectasia	
	V		Spinal venous drainage	Progressive myelopathy in 50%

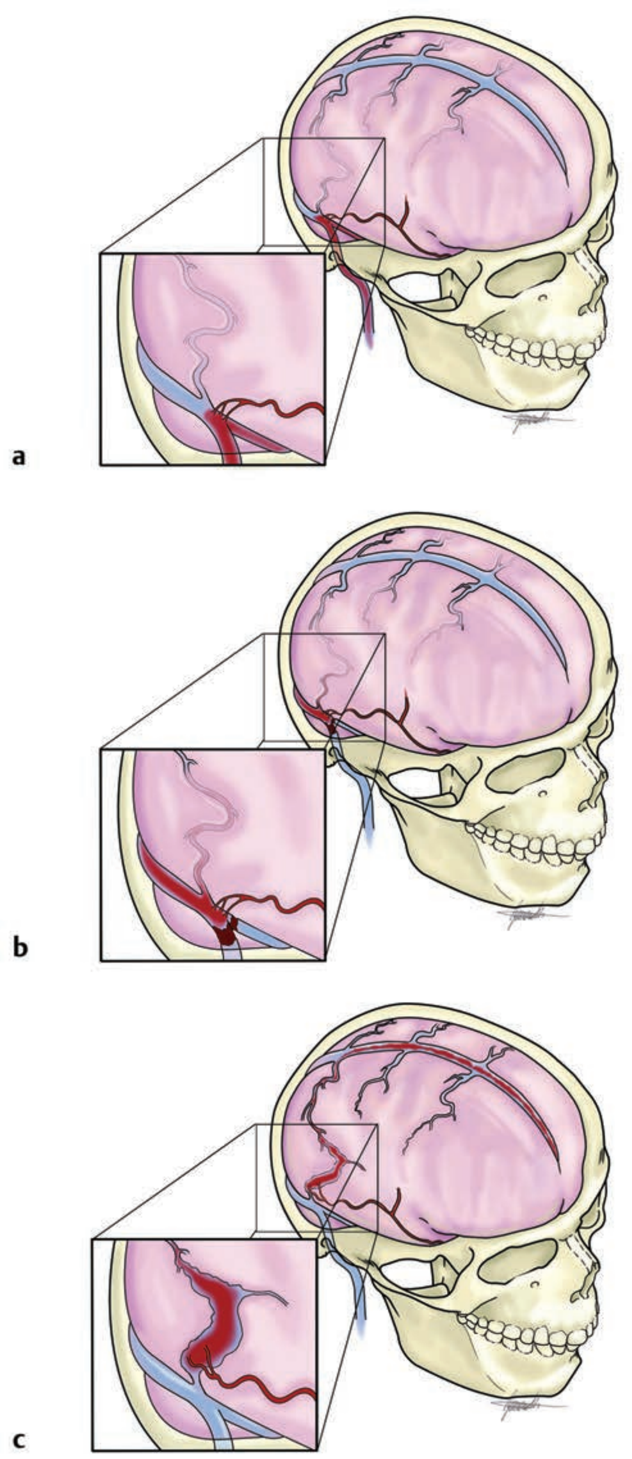

Fig. 32.1 Transverse-sigmoid junction DAVF with middle meningeal artery supply. **(a)** Borden type I with anterograde flow in sigmoid sinus. **(b)** Borden type II with occlusion of sigmoid and superior petrosal sinuses, retrograde flow into transverse sinus and cortical venous reflux. **(c)** Borden type III with cortical venous reflux plus ectasia and no sinus involvement.

The clinical presentation and natural history of DAVF have been studied according to the characteristics of venous drainage.[1,7] In Borden type I (equivalent to Cognard types I and IIa), most patients with transverse/sigmoid sinus DAVF present with pulsatile tinnitus. The incidence of intracranial hemorrhage and progressive neurological deficit is about 2%.[1] These lesions are thus considered benign and may undergo spontaneous resolution.[2] Progression to Borden types II or III is rare, and has been reported in 2 to 3% of cases after a mean follow-up of 7 years.[7,9] Borden types II and III are regarded as aggressive lesions. They are associated with a clinical presentation of intracranial hemorrhage and neurological deficits in 39 and 79% of Borden II and III patients, respectively.[4] Based on natural history studies, the annual hemorrhage risk for such DAVF is 8.1%, and risk of nonhemorrhagic neurological deficit is 6.9%.[10] In patients who have presented with a previous intracranial hemorrhage from Borden types II and III DAVF, there is a 20 to 35% risk of rebleeding within a 2-week period.[11] The putative mechanism for this is venous hypertension from retrograde leptomeningeal flow, leading to venous congestion, cerebral edema, infarction and intracranial hemorrhage.[2,7,8]

32.3 Treatment for DAVF

32.3.1 Indications for treatment

Based on the more benign natural history, Borden type I lesions can be managed in most instances with observational treatment.[2,9] In some cases, however, treatment may be offered as palliation for symptomatic pulsatile tinnitus.[7,9] In Borden types II and III, urgent treatment should be offered soon after the diagnosis is made.[10,11] The objective of treatment is to completely eliminate the cortical venous reflux, which may be achieved through division or obliteration of the arterialized leptomeningeal draining vein.[5,8,11]

32.3.2 Treatment modalities

Available treatment modalities include endovascular embolization, microsurgery, and radiotherapy, whether as single or combination therapies.[2,7,10,12] Embolization techniques are the first-line treatment.[3] Embolization may be performed through a transvenous or transarterial approach, or even through both approaches. Transvenous embolization and obliteration of the sinus and arterialized vein/venous pouch is the preferred treatment for Borden II or Cognard IIb lesions, as it offers the best chance for cure.[1] The affected sinus and arterialized venous pouch can be embolized with platinum coils or liquid embolic agents, such as n-butyl cyanoacrylate (Cordis, Miami Lakes, FL) and ethylene vinyl alcohol copolymer (Onyx, ev3, Irvine, CA, ▶ Fig. 32.2**b,** ▶ Fig. 32.3**b**).[7,8] It is important to ensure that the involved sinus is not responsible for normal cortical venous drainage.[7] In Borden type III DAVF, the venous sinus is not involved and may be trapped or thrombosed. The objective of treatment is to obliterate the cortical venous reflux, which is occasionally successful with transvenous embolization if a catheter can be manipulated into the arterialized venous pouch through the thrombosed or trapped segment of the sinus.[12] Most of these cases, however, may require surgical disconnection.

Transarterial treatment may be offered when transvenous embolization is not suitable due to venous access difficulties, risks of venous, or when further evaluation of high-flow fistulous lesions

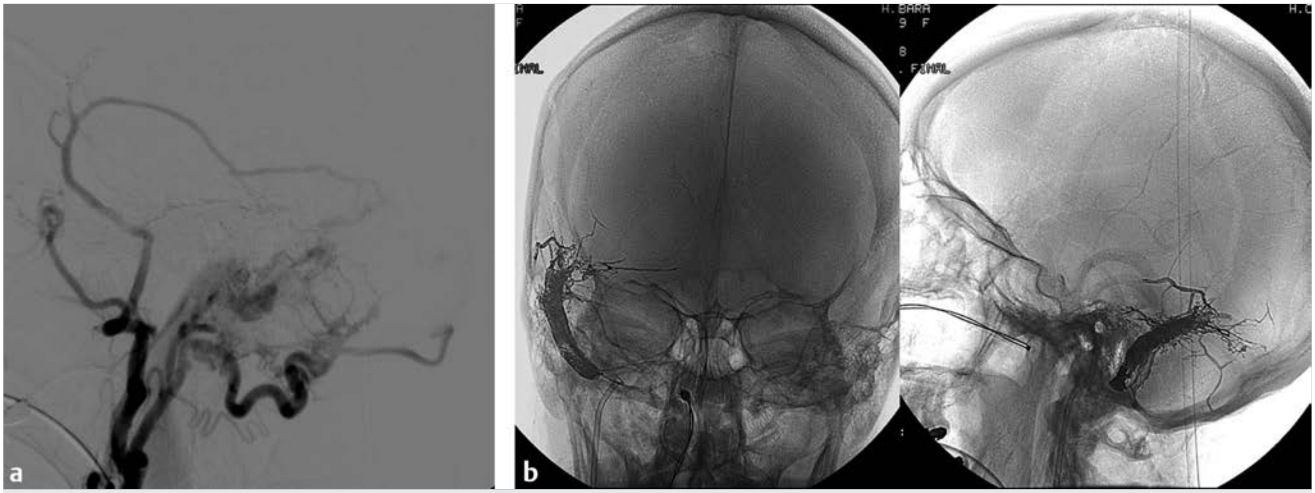

Fig. 32.2 (a) External carotid arteriogram of Borden type I transverse sinus DAVF. **(b)** Transarterial embolization with Onyx and transvenous embolization with coils.

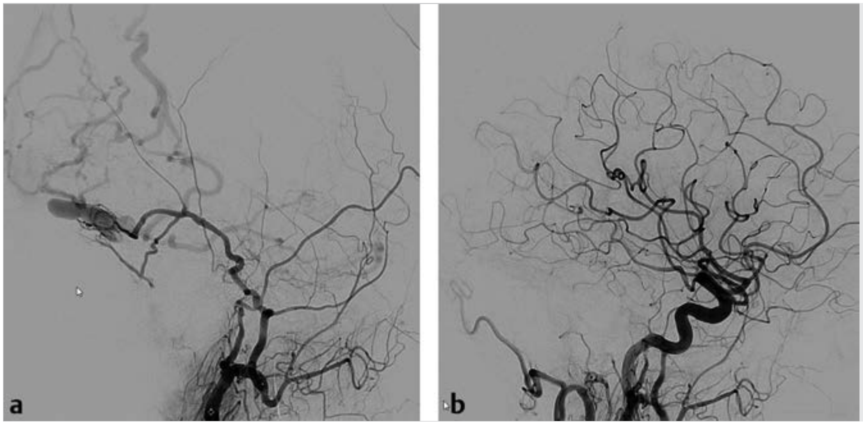

Fig. 32.3 (a) External carotid arteriogram of Borden type II DAVF with cortical venous reflux. **(b)** Angiogram following transarterial embolization with Onyx.

is necessary through selective arterial embolizations.[7,8] The objective is to penetrate the fistulous point via the arterial feeders, either with liquid embolic agents or particles.[2,7,12] In general, fistula cure with transarterial approach is low because multiple feeders may be involved, feeding arteries may not be accessible due to tortuosity despite the use of microcatheters or there may be limited penetration through intraosseous branches with embolic materials[12]; also, recanalization and partial treatment lead to DAVF recurrence. Thus, transarterial tends to be used more frequently for symptom palliation, rather than curative intent.

Surgery is an alternative option when embolization is not possible. In Borden III DAVF, only disconnection of the arterialized draining vein at the dural fistulous point is necessary.[13] Similarly, in Borden II DAVF, disconnection of the arterialized draining vein may be performed, thus converting it to a Borden I DAVF. Alternatively, the affected dural sinus may be excised or packed surgically.[13]

Stereotactic radiosurgery is another option that is infrequently offered. The main disadvantage is the lag period of at least 2 years for DAVF occlusion to occur.[8,10,11]

32.4 Surgery for Transverse and Sigmoid DAVF

32.4.1 Preoperative Assessment and Preparation

This requires a 6-vessel digital subtraction angiogram, which includes bilateral internal carotid, external carotid, and vertebral artery injections.[7,13] The arterial supply of DAVF and its fistulous connections (whether single or multiple), direction of venous sinus drainage, and the presence and location of retrograde cortical venous reflux or ectasia should be evaluated. It is important to determine the pattern of normal cortical venous drainage, as sacrifice of anterograde draining veins or functioning sinus may lead to venous infarction.[7,8] Preoperative CT and MRI with angiography are useful to localize the affected sinus or arterialized draining vein, especially when the images are merged for intraoperative stereotactic navigation.[7]

Massive blood loss may occur, even from the craniotomy opening, and anesthesia should be prepared for rapid blood transfusion requirements. Based on the degree of arteriovenous shunting on angiography, transarterial embolization may be considered to minimize blood loss prior to surgery.[5,7,8,13]

32.5 Operative Technique

32.5.1 Disconnection of Cortical Venous Reflux (Video 32.1)

Our preferred surgical technique for treatment of transverse and sigmoid DAVF is disconnection of the arterialized draining vein to eliminate cortical venous reflux.

Positioning

Typically, the patient is positioned in the lateral decubitus position (or park-bench position), and the head is turned and fixed such that the occipital bone between the inion and asterion over the ipsilateral transverse sinus is parallel to the floor. Alternatively, the patient may be prone if infratentorial access is required.

Craniotomy

The craniotomy is planned and tailored according to the exact site of the fistula, ensuring the arterialized draining vein and the adjacent region of the transverse sinus and tentorium are exposed. This involves an occipital craniotomy for retrograde cortical venous reflux in the supratentorial compartment, and exposure of the infratentorial dura is required if there is venous reflux toward the cerebellum. Intraoperative stereotactic navigation is a useful adjunct to localize the site of cortical venous reflux or venous pouch.[3,7]

During skin incision and elevation of the scalp flap, the occipital artery may be cauterized, thereby reducing the blood supply to the DAVF.[3] Burr holes are placed for the craniotomy and elevation of a free bone flap, with bone bleeding controlled with bone wax.[5,7] Major hemorrhage may occur during this stage of the operation, but may be reduced with preoperative arterial embolization of external carotid artery feeders. Meningeal arteries are coagulated or occluded with hemostatic clips during dural opening, and the dural flap is reflected along the transverse sinus.[5,7]

Disconnection of Retrograde Leptomeningeal Vein

The intradural dissection is performed with the intraoperative microscope and leptomeningeal veins are exposed as they drain into the sinus or tentorium. Arterialized leptomeningeal veins do not drain blood from functional brain and can be obviously distinguished from normal cortical draining veins in most instances, since arterialized veins will appear “red” (▶ Fig. 32.4**c**) and normal cortical veins “blue.”[13] This may be further confirmed with the use of intraoperative indocyanine green (ICG) angiography, which can reveal the direction and timing of flow through the leptomeningeal vessels (▶ Fig. 32.4**d**). Arterialized veins are coagulated with the bipolar forceps or clipped with a permanent aneurysm clip prior to division at the entrance into the dural venous sinus, or as close to the fistula site as possible.[2,5] Important caveats include: (1) ensuring that the arterialized draining vein is adequately coagulated before division; (2) all arterialized leptomeningeal veins are disconnected with a thorough inspection of the adjacent dura; and (3) nonarterialized cortical veins are not sacrificed.[7,8] Unlike brain arteriovenous malformations, in which draining veins immediately change to blue color when all AVM shunts are disconnected, cortical veins of DAVF may require more time for color change. A second ICG angiogram may be useful to confirm the absence of retrograde flow once the DAVF has been disconnected.[7] Closure of the wound then proceeds in the usual manner with dural closure and replacement of the bone flap.

32.5.2 Skeletonization and Packing of Sinus

In cases involving multiple fistulous sites, skeletonization of the venous sinus may be performed by coagulating the dural borders above, below, and medial to the sinus, with or without incision through the coagulated dural borders.[2,3] Thus, a craniotomy allowing access to the occipital supratentorial and infratentorial supracerebellar compartments is required to expose and preserve the entire transverse sinus.[13]

Another possible operation is to expose a small portion of the sinus for direct intraoperative catheterization.[3,12] This technique may be used when the usual transvenous approaches are not available due to steno-occlusive disease of the dural venous sinuses. The sinus can be directly packed and obliterated with coils, or other thrombogenic materials, such as Gelfoam (Pfizer Inc., New York, NY) or silk sutures.

32.5.3 Postoperative Management and Treatment Outcomes

A postoperative catheter angiogram is advised to ensure that the cortical venous reflux has been completely eliminated.[8,13] Any DAVF with residual cortical venous reflux should be considered for further treatment due to the high risk of hemorrhage, with either endovascular embolization or stereotactic radiosurgery.[3] Long-term follow-up should be provided to detect recurrences, with repeated vascular imaging, such as catheter or time-resolved MR angiography.

Treatment outcomes are mostly correlated with the initial presentation of DAVF, rather than treatment related. Obliteration rates for DAVF between 91 and 100% have been reported in various case series.[3,8,13] Morbidity and mortality of up to 17% have been reported, including venous infarction, postoperative hematoma and death.[3,4]

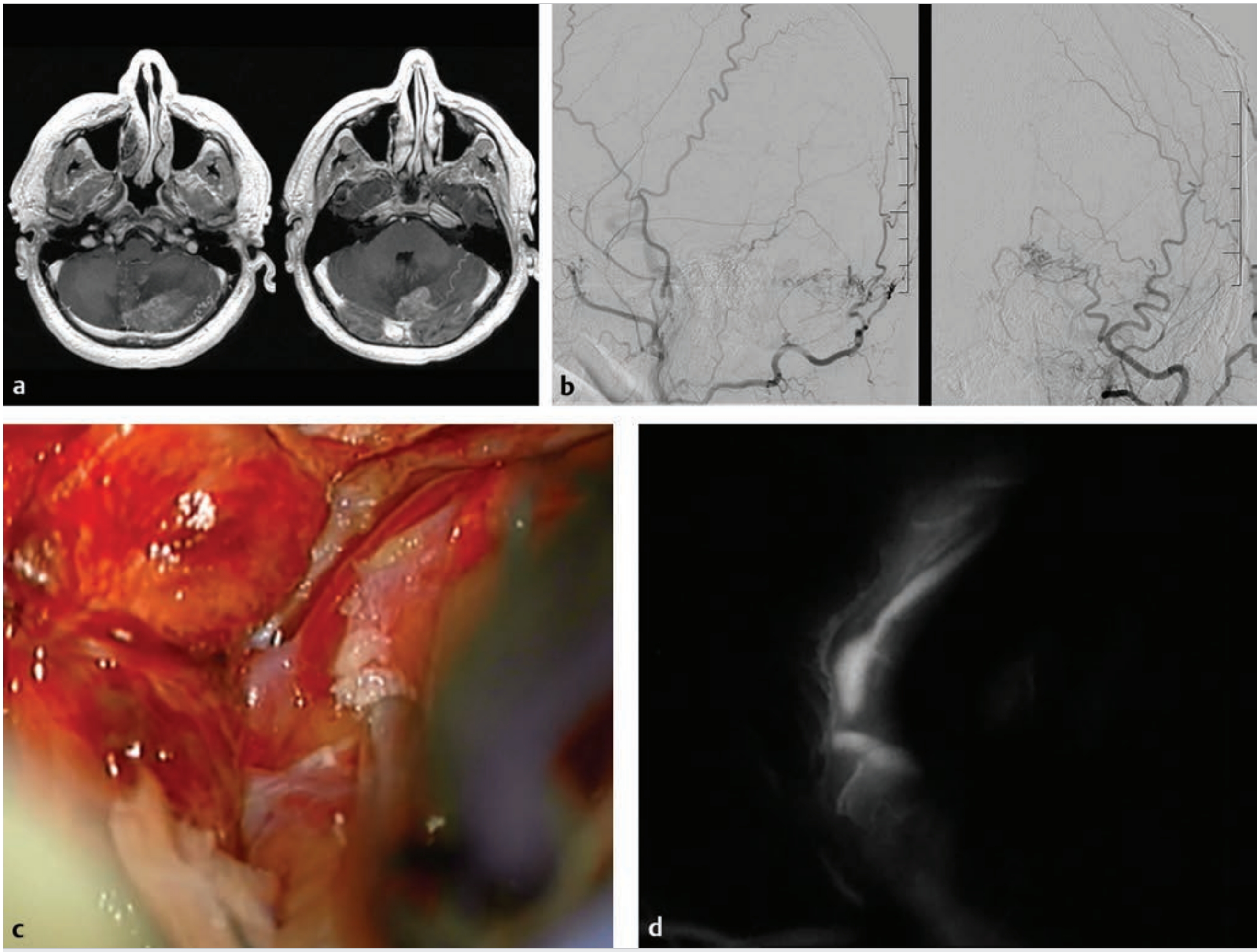

Fig. 32.4 **(a)** Axial T1-weighted MRI with gadolinium of Borden type III DAVF with contrast enhancement and prominent blood vessels. **(b)** Preoperative lateral (left) and anteroposterior (right) external carotid arteriogram with transosseous supply from left occipital artery and venous reflux into superior vermian vein. **(c)** and **(d)** Intraoperative photo and ICG angiogram of "red" arterialized vein and venous pouch before disconnection of cortical venous reflux.

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33 Tentorial and Posterior Fossa Dural Arteriovenous Fistulas

Tyler S. Cole, Martin J. Rutkowski, Peter Nakaji, and Michael T. Lawton

Abstract

Dural arteriovenous fistulas (DAVFs) are abnormal connections between an artery or arteries that supply the dura mater and a vein or venous sinus contained within the leaflets of dura. Aberrant hemodynamics make DAVFs susceptible to hemorrhage, often with an annual rupture risk that is three to five times greater than that of brain arteriovenous malformations (AVMs); however, the incidence is one-tenth that of AVMs. Although most DAVFs can be treated with neurointerventional techniques, posterior fossa and tentorial DAVFs tend to be high-risk for hemorrhage and can fail endovascular embolization or are not amenable to endovascular therapy due to anatomical constraints. Although headache and tinnitus are common presentations, in the posterior fossa the variety of presenting symptoms can be wide and includes double vision, visual obscuration, balance issues, or other focal neurologic deficits related to cranial nerve, brainstem, and cerebellar involvement. This subset of DAVFs is particularly susceptible to rupture and disproportionately present with hemorrhage and progressive neurologic deficit. We outline the six major types of tentorial DAVFs and three major types of posterior fossa DAVFs and discuss surgical techniques compared to endovascular treatment, as well as operative nuances for those fistulas treated with open surgery.

Keywords: dural arteriovenous fistula, endovascular embolization, hemorrhage, rupture, microsurgery

33.1 Introduction

Dural arteriovenous fistulas (DAVFs) represent abnormal junctions between arteries and veins or venous sinuses within the dural leaflets. Aberrant hemodynamics make DAVFs susceptible to hemorrhage, often with an annual rupture risk that is three to five times greater than that of brain arteriovenous malformations (AVMs); however, the incidence is one-tenth that of AVMs. These lesions are believed to be acquired, not congenital, with a pathogenesis that is increasingly understood. They can be treated safely and effectively with endovascular, microsurgical, and combined techniques. However, they require a keen diagnostic sense to plan optimal interventions. The majority of DAVFs can be managed and cured through an endovascular approach; nonetheless, posterior fossa and tentorial DAVFs tend to be high-risk for hemorrhage and often fail endovascular embolization, or they are not amenable to endovascular therapy due to anatomical constraints. Although headache and tinnitus are common presentations, in the posterior fossa the variety of presenting symptoms can be wide and includes double vision, visual obscuration, and balance issues or other focal neurologic deficits related to cranial nerve, brainstem, and cerebellar involvement.[1,2,3,4,5,6,7] DAVFs located in the cortical venous drainage or in the posterior fossa are independent predictors of hemorrhage. DAVF should be suspected in patients with nontraumatic intracranial hemorrhage with a subarachnoid component.[7]

33.2 DAVF Classification, Natural History, and Indications for Treatment

The Borden classification is one of several DAVF classification systems that have been proposed. The Borden classification is the most clinically useful system and unifies and simplifies the other classification systems; it has become the accepted classification system.[8] Type I DAVFs drain in an anterograde direction into the associated dural venous sinus or meningeal veins. They almost never hemorrhage or produce neurologic deficits, and they are treated only if they become symptomatic. A small number can evolve into type II or III lesions so imaging surveillance may be indicated. Type I DAVFs are treated with transarterial embolization or surgical skeletonization of the venous sinuses if venous drainage needs to be preserved.

Type II DAVFs similarly not only drain into dural venous sinuses or meningeal veins but also drain in a retrograde direction into cortical veins. Type III DAVFs drain exclusively into cortical veins, without venous sinus or meningeal venous drainage. Borden types II and III DAVFs are associated with a risk of hemorrhage and progressive neurologic deficits. Treatment is indicated in patients with these types of DAVFs. Risk factors for hemorrhage include cortical venous drainage, posterior fossa location, and history of hemorrhage.[7,9] Type II DAVFs are treated by interruption of the arterialized draining cortical vein and occlusion or excision of the venous sinus. Type III DAVFs can be treated by disruption of the arterialized draining cortical vein.

Resection of DAVFs is not required as with brain AVMs. The draining vein may be occluded using an endovascular method, without resecting the dura containing the DAVF.

33.3 Tentorial DAVFs

Tentorial DAVFs are rare and dangerous lesions.[8,10,11,12,13,14,15] A meta-analysis of patients ($n = 377$) with tentorial, transverse-sigmoid, and cavernous DAVFs showed that patients with tentorial DAVFs had the highest hemorrhage rate or clinical neurologic decline (31/32, 97%).[10] Tentorial DAVFs are challenging to treat with endovascular therapy and should be decisively treated.[13] They have an extensive arterial supply that involves the meningeal arteries from the internal carotid artery and vertebral artery, which are more difficult to embolize and carry greater risk than DAVFs supplied by the external carotid artery. Additionally, tentorial DAVFs frequently drain into subarachnoid veins instead of to their associated sinus (Borden type III), which precludes a transvenous approach.[8] Therefore, the management of tentorial DAVFs typically requires microsurgical intervention, in contrast to most other DAVFs.[12,16,17,18,19,20,21,22]

Tentorial DAVFs may be categorized into six types, based on anatomical location, dural base, related venous sinus, and type of venous drainage (▶ Fig. 33.1). Type I galenic DAVFs may be found in the midline at the posterior border of the tentorial incisura and in association with the vein of Galen as it enters the anterior falcotentorial junction. They may have either supratentorial or infratentorial drainage or both (▶ Fig. 33.2). Type II DAVFs are located in the midline along the falcotentorial junction. They are associated with the straight sinus and drain into veins on the undersurface of the tentorium. Type III DAVFs are located in the midline at the posterior border of the falcotentorial junction, and they are associated with the torcula and have supratentorial venous drainage. Type IV DAVFs may be found in the body of the tentorium in association with the tentorial sinus and drain supratentorially into the occipital veins. Type V DAVFs are located laterally where the tentorium joins the dura of the middle cranial fossa. They are associated with the superior petrosal sinus and drain infratentorially into the petrosal vein and its tributaries. Type VI incisural DAVFs may be found along the free edge of the tentorium and are not associated with a venous sinus. They drain into the supratentorial veins in and around the ambient cistern.

33.3.1 Galenic DAVFs

Of the six types of tentorial DAVFs, the galenic type tends to be the most difficult to address. The area around the vein of Galen is particularly deep, and the natural barriers where the falx and tentorium meet can be awkward to navigate. There is no typical direction of arterial inflow. Furthermore, the dilated, tortuous venous architecture often makes the fistulous outflow hard to determine. The combination of surgical depth and complex inflow and outflow necessitates a wide, panoramic exposure that only a torcular craniotomy with an interhemispheric approach can provide (▶ Fig. 33.3, **Video 33.1**).[23] With the patient positioned laterally, this approach provides excellent exposure of the torcula, bilateral transverse sinuses, and superior sagittal sinus. After exposing and entering the dura, a wide interhemispheric split is obtained by gravity retraction of the lower occipital lobe, without the use of a retractor (▶ Fig. 33.4). Skeletonizing the straight sinus by cutting the falx superior to the straight sinus and the tentorium bilateral to the straight sinus allows views on both sides of the falx and the tentorium. The ambient and quadrigeminal cisterns are readily viewed. If the surgeon prefers, the patient may be positioned for a supracerebellar-infratentorial approach to these lesions. The main advantage of this approach is that, in the case of fistulas that drain inferiorly to cerebellar veins, the surgeon is positioned on the appropriate side of the tentorium for excellent venous exposure. Tentorial incisions can then be made to expose the relevant anatomy more superiorly. The surgeon should consider that the steep slope of the tentorium medially near the vein of Galen can potentially limit the view of critical structures, particularly when the venous anatomy is dilated and tortuous.

Another benefit of skeletonization is concurrent dearterialization of the lesion as the ECA and tentorial feeders are eliminated laterally and the middle meningeal and falcine feeders are eliminated superiorly. The occipital artery supply is eliminated by the craniotomy and dural exposure. If posterior meningeal artery feeders are present, they can be identified and coagulated during dural exposure. However, the primary goal is accomplished when the venous drainage is obliterated. Exposure and skeletonization is mainly a means to better understand and completely visualize the venous anatomy, with the benefit of arterial occlusion.

After careful and delicate dissection of the outflow veins from the fistula, clipping or coagulation of the veins is then performed. The internal cerebral veins are prominent in the posterior view provided by a torcular interhemispheric approach; however, they are rarely the target of occlusion. More commonly, the precentral cerebellar vein and the basal vein of Rosenthal are the targets of occlusion, but they tend to be difficult to occlude. When identifying the basal vein of Rosenthal, a lateral and inferior trajectory can be taken into the ambient

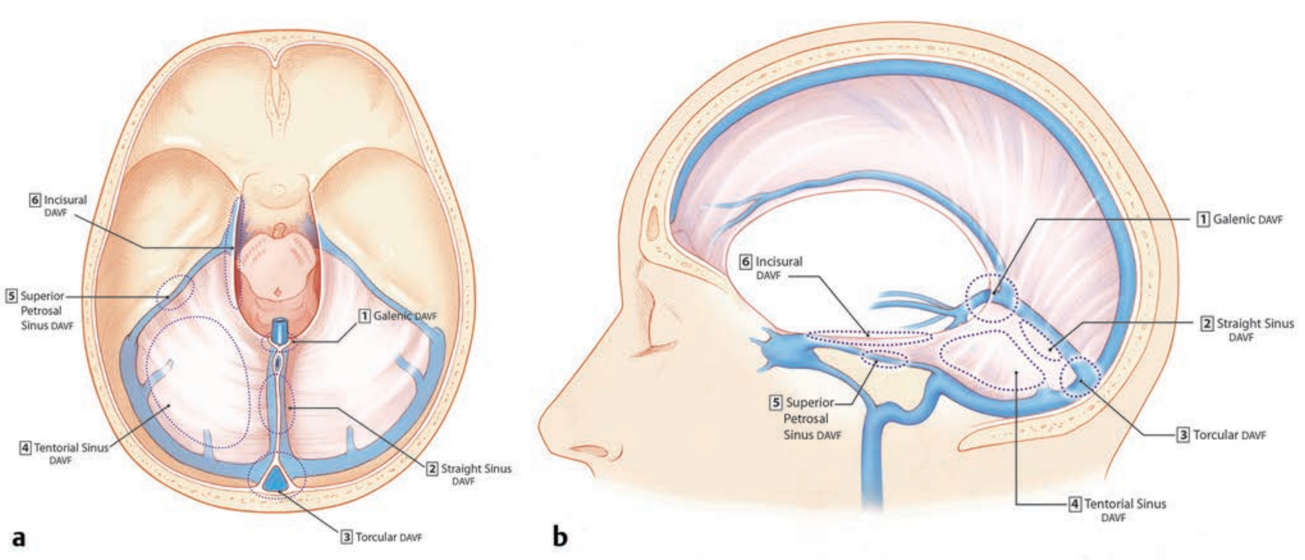

Fig. 33.1 Illustration of the six types of tentorial dural arteriovenous fistulas. **(a)** Axial view and **(b)** sagittal view. DAVF, dural arteriovenous fistula. (Reproduced with permission from Lawton MT, Sanchez-Mejia RO, Pham D, et al. Tentorial dural arteriovenous fistulae: operative strategies and microsurgical results for six types. Neurosurgery 2008;62:110–124, discussion 124–125.)

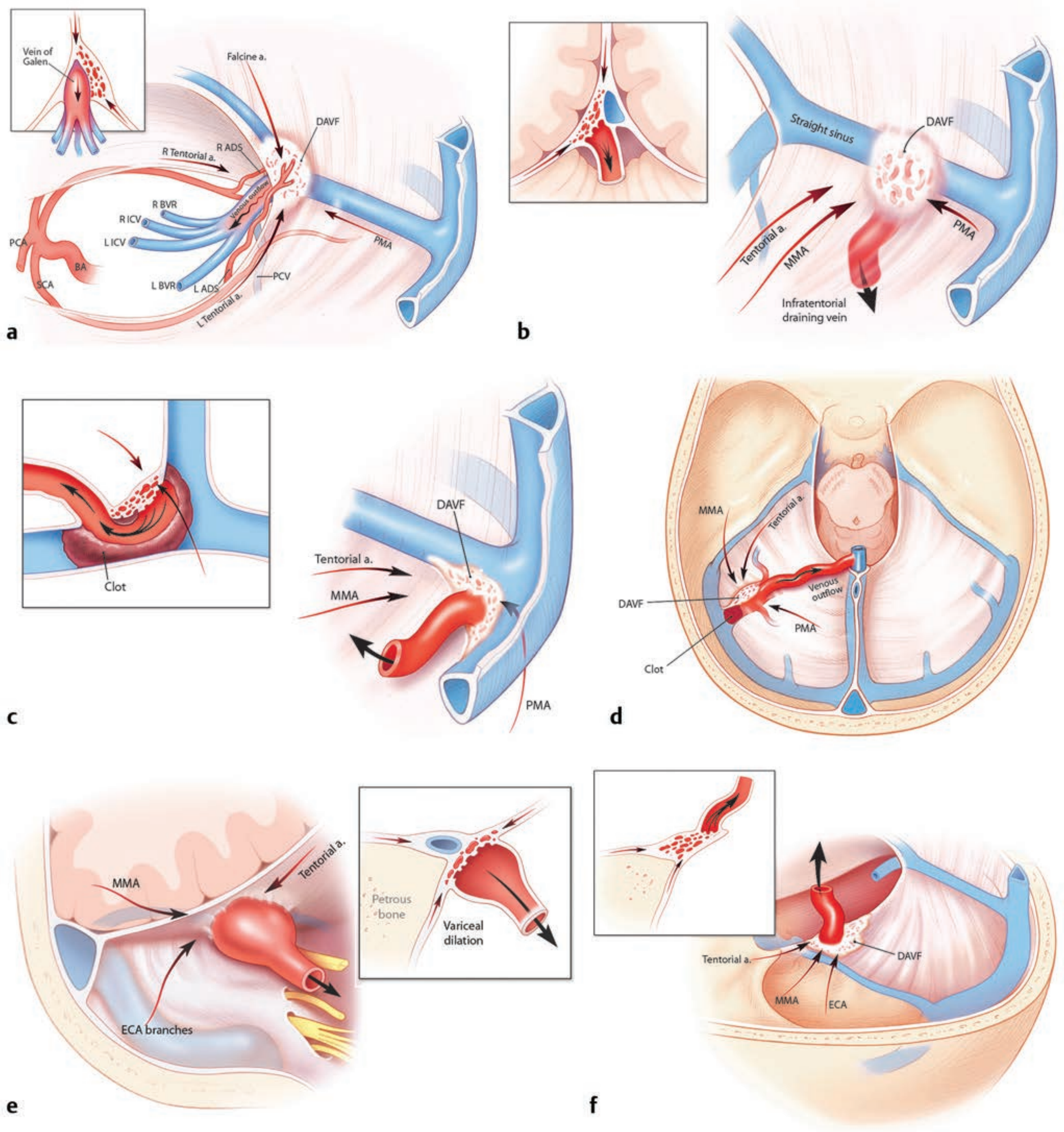
Vein of Galen
Falcine a.
DAVF
R ADS
R Tentorial a.
Venous outflow
R BVR
R ICV
L ICV
PCA
BA
SCA
L BVR
L ADS
PCV
PMA
L Tentorial a.
a
Straight sinus
DAVF
Tentorial a.
MMA
PMA
Infratentorial draining vein
b
Clot
DAVF
Tentorial a.
MMA
PMA
c
MMA
Tentorial a.
Venous outflow
DAVF
PMA
Clot
d
MMA
Tentorial a.
ECA branches
Petrous bone
Variceal dilation
e
Tentorial a.
DAVF
MMA
ECA
f

Fig. 33.2 Anatomy of tentorial DAVFs by type. **(a)** Galenic DAVF (type I). **(b)** Straight sinus DAVF (type II). **(c)** Torcular DAVF (type III). **(d)** Tentorial sinus DAVF (type IV). **(e)** Superior petrosal sinus DAVF (type V). **(f)** Incisural DAVF (type VI). (a., artery; ADS, artery of Davidoff and Schecter; BA, basilar artery; BVR, basal vein of Rosenthal; DAVF, dural arteriovenous fistula; ECA, external carotid artery; ICV, internal cerebral vein; L, left; MMA, middle meningeal artery; PCA, posterior cerebral artery; PCV, precentral cerebellar vein; PMA, posterior meningeal artery; R, right; SCA, superior cerebellar artery. (Reproduced with permission from Lawton MT, Sanchez-Mejia RO, Pham D, et al. Tentorial dural arteriovenous fistulae: operative strategies and microsurgical results for six types. Neurosurgery 2008;62:110–124, discussion 124–125.)

cistern. The precentral cerebellar vein is typically located out of view in the surgical corridor inferior to the junction of the falx and tentorium, which after skeletonization can be mobilized easily to identify the cerebellar veins. If an identified vein has a red color and dilated morphology, this appearance indicates fistulous drainage. Blue-colored veins rarely drain pathologic regions. Visual inspection of the vascular anatomy intraoperatively does not always reveal an obvious relationship between the fistula-containing dura and the venous outflow that has been arterialized.

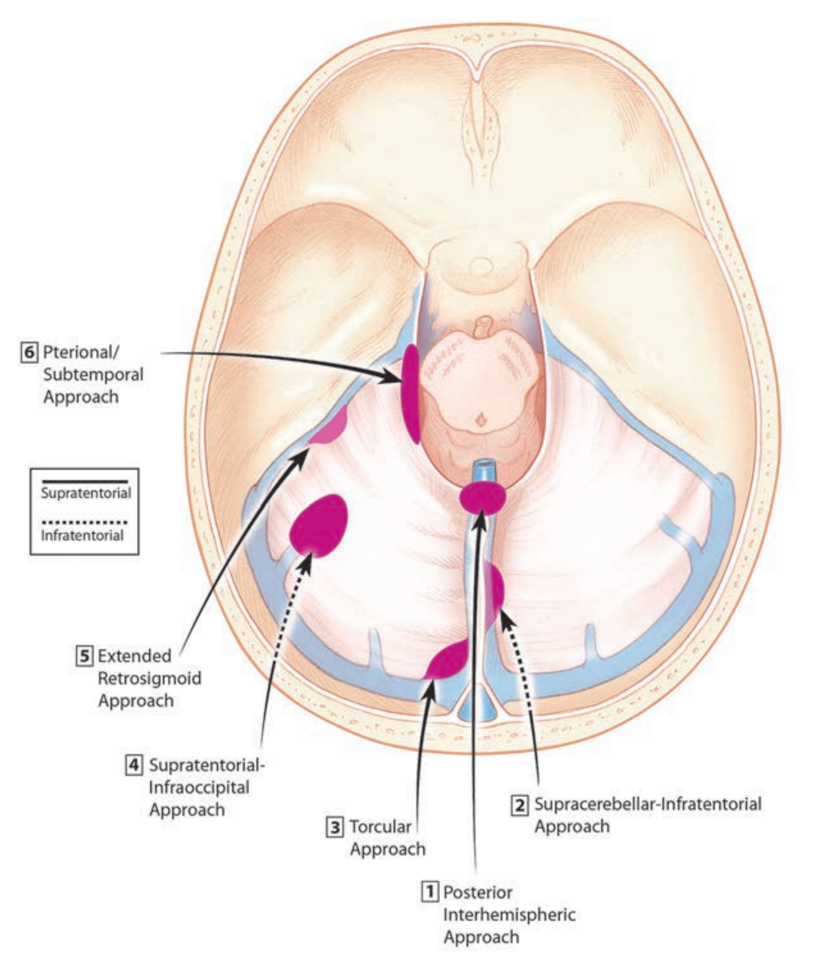
6 Pterional/ Subtemporal Approach
Supratentorial
Infratentorial
5 Extended Retrosigmoid Approach
4 Supratentorial-Infraoccipital Approach
3 Torcular Approach
1 Posterior Interhemispheric Approach
2 Supracerebellar-Infratentorial Approach

Fig. 33.3 Summary of surgical approaches to tentorial dural arteriovenous fistulas. (Reproduced with permission from Lawton MT, Sanchez-Mejia RO, Pham D, et al. Tentorial dural arteriovenous fistulae: operative strategies and microsurgical results for six types. Neurosurgery 2008;62:110–124, discussion 124–125.)

The patency of the straight sinus should be determined by preoperative angiography, as this influences venous occlusion. Most often, galenic DAVFs tend to be Borden type III, draining in a retrograde direction into the vein of Galen or its smaller branches. If the straight sinus is occluded, a clip can be placed directly on the galenic trunk to address the fistulous outflow. The dissection involved in this situation is not extensive and is anatomically straightforward. However, not all straight sinuses are obstructed in these patients. Occasionally, on the venous phase of angiography, antegrade flow is observed in the straight sinus. If this is the case, the patent vein of Galen should be preserved, with care taken to clip or coagulate only the tributary branches of the vein of Galen without interrupting its trunk. If the galenic tributary veins are clearly blue and nonarterialized, these veins may be left to continue draining the deep cerebral circulation to maintain antegrade outflow from normal tissue. (Treatment of galenic DAVFs is discussed in greater detail in Chapter 26.)

33.3.2 Straight Sinus DAVFs

Surgical treatment of straight sinus DAVFs is often performed with the patient in the sitting position via the supracerebellar infratentorial corridor. These DAVFs are not as deep as galenic DAVFs and typically drain via a single vein. There usually is no need to dissect and skeletonize the venous anatomy (▶ Fig. 33.2). With the patient in the sitting position, gravity retraction of the cerebellum opens the natural subarachnoid plane to expose the fistula. Even with patients who present with a subarachnoid or an intraparenchymal hemorrhage causing cerebellar edema and narrowing of the surgical corridor, this approach is often adequate (▶ Fig. 33.5). Depending on the experience of the surgeon, the patient may be positioned in a semiprone park bench position for the supracerebellar infratentorial approach. However, retractors are required for a good exposure. The primary disadvantage of retractor use is the risk of venous avulsion. If the avulsed vein is related to the fistula, extensive bleeding may occur outside the immediate surgical view.

The prone position is often preferred for the supracerebellar intratentorial approach because it allows the surgeon to sit while operating. The surgeon may also sit during procedures with the patient in the sitting position. The back of the patient should approach vertical while the head is flexed to align the level of the tentorium almost horizontally. The surgeon can then sit on a stool in a free-standing, arm-embracing position that supports the elbows, arms, and hands at shoulder level. This setup relaxes the surgeon's arms and stabilizes the surgeon's hands. Since dissection and fistula occlusion are procedures of limited duration, the surgeon can easily tolerate this awkward arm position.

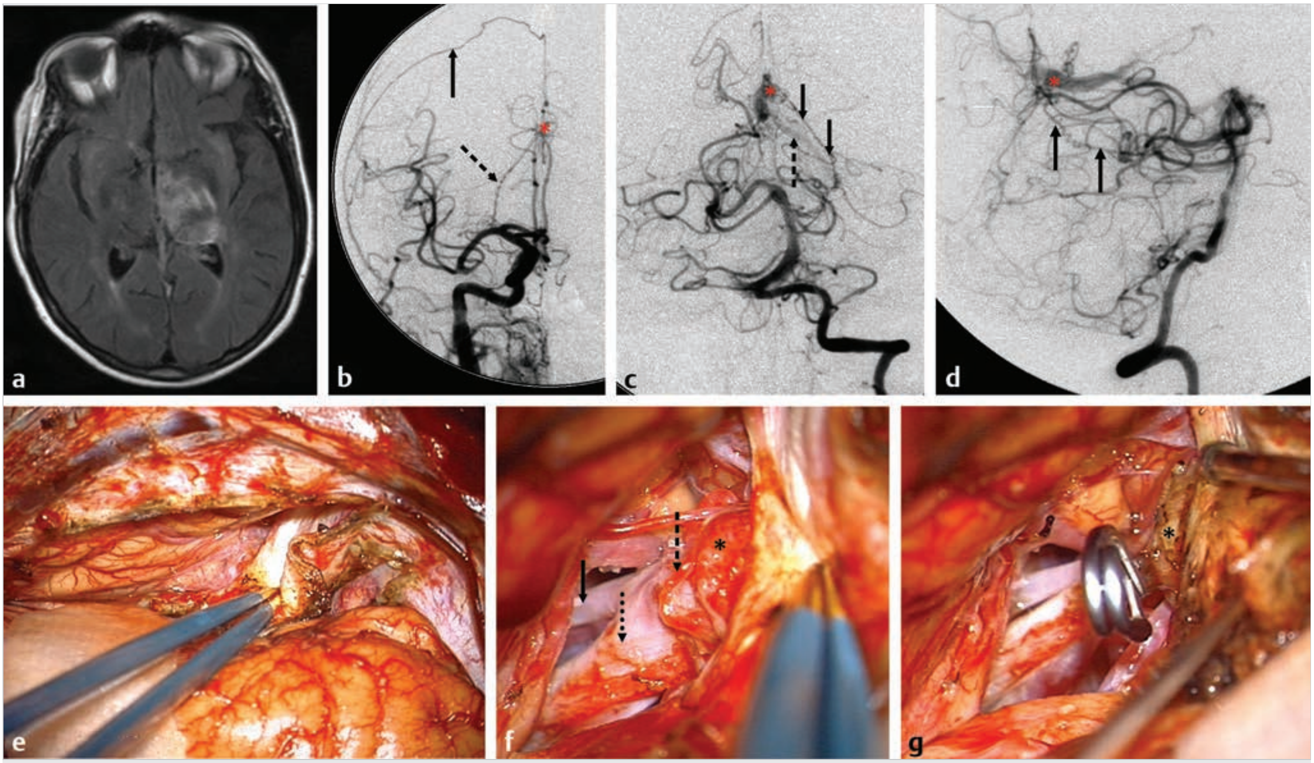

Fig. 33.4 Galenic dural arteriovenous fistulas (type I). **(a)** Axial brain magnetic resonance imaging (fluid-attenuated inversion recovery sequence) showed increased signal in the left thalamus caused by retrograde venous drainage from the fistula to the vein of Galen, left basal vein of Rosenthal, and left internal cerebral vein. **(b)** Right internal carotid artery angiogram (anteroposterior view) demonstrated arterial supply to the fistula (*red asterisk*) from the right tentorial artery (*dashed arrow*) and middle meningeal artery (*solid arrow*). Left vertebral artery angiograms (**c**, anteroposterior view; **d**, lateral view) demonstrated an arterial supply to the fistula (*red asterisk*) from the artery of Davidoff and Schechter (*solid arrows*) and drainage into the vein of Galen and left basal vein of Rosenthal (**c,** *dashed arrow*). **(e)** Intraoperative photograph demonstrates the posterior interhemispheric approach, with the patient positioned laterally (left side down), with gravity retraction of the left occipital lobe, and with transection of the left tentorium and falx (at tips of bipolar forceps) to widen the exposure. **(f)** Reflection of the dura at the falcotentorial junction with the bipolar forceps provides visualization of the fistula (*black asterisk*) and the vein of Galen complex (right basal vein of Rosenthal, *solid arrow*; right internal cerebral vein, *dotted arrow*; and artery of Davidoff and Schechter, *dashed arrow*). **(g)** The straight sinus was already occluded, so the fistula (*black asterisk*) was interrupted with a clip placed on the vein of Galen as it exited the fistula. (Reproduced with permission from Lawton MT, Sanchez-Mejia RO, Pham D, et al. Tentorial dural arteriovenous fistulae: operative strategies and microsurgical results for six types. Neurosurgery 2008;62:110–124, discussion 124–125.)

For the supracerebellar-infratentorial approach, the suboccipital craniotomy spans from just superior to the transverse sinuses and torcula to just short of the foramen magnum. It is important to expose the torcula so as to eliminate the bony edge that would otherwise obscure the infratentorial plane. Surgeons must be mindful that, during exposure of the torcula, venous sinus injury and dural tears can be dangerous, particularly when the patient is in the sitting position, as lowering the head is difficult in the case of suspected air embolus. In older patients with a dura adherent to the cranium during exposure, it may be wise to avoid crossing the sinus with the craniotome. Instead, the surgeon may consider performing a suboccipital craniotomy first and then commence dural dissection under direct visualization along the inner table of the skull. An alternate approach is to drill away the bone overlying the sinuses with a diamond tip drill until the inferior margins of the venous sinuses are visualized. With the torcula exposed, the dural flap is raised with the anchoring edge along the transverse sinuses. There should be sufficient tension to elevate the torcula. The arterialized vein providing fistulous outflow can then be located on the surface of the cerebellum by its redness and followed back to the fistula. It may also be possible to see the arterialized vein exiting the dura in the subarachnoid space. The draining vein can be identified by its relatively thicker wall and distinctive white color with red vasa vasorum. A clip should be placed on the draining vein as it exits the dura of the tentorium, which is then followed by coagulation and cutting.

33.3.3 Torcular DAVFs

Among torcular DAVFs, the difference in treatment strategies between Borden types II and III torcular DAVFs should be discussed. Type III fistulas that drain solely into adjacent veins are treated in a simple fashion by clipping the arterialized veins as they leave the sinus. A torcular craniotomy is used to expose these superficial fistulas with the patient in a prone position, and typically minimal to no subarachnoid dissection is needed. In contrast, treating type II torcular fistulas is more difficult than treating type III fistulas because they drain into torcular sinuses and nearby veins. Although the arterialized

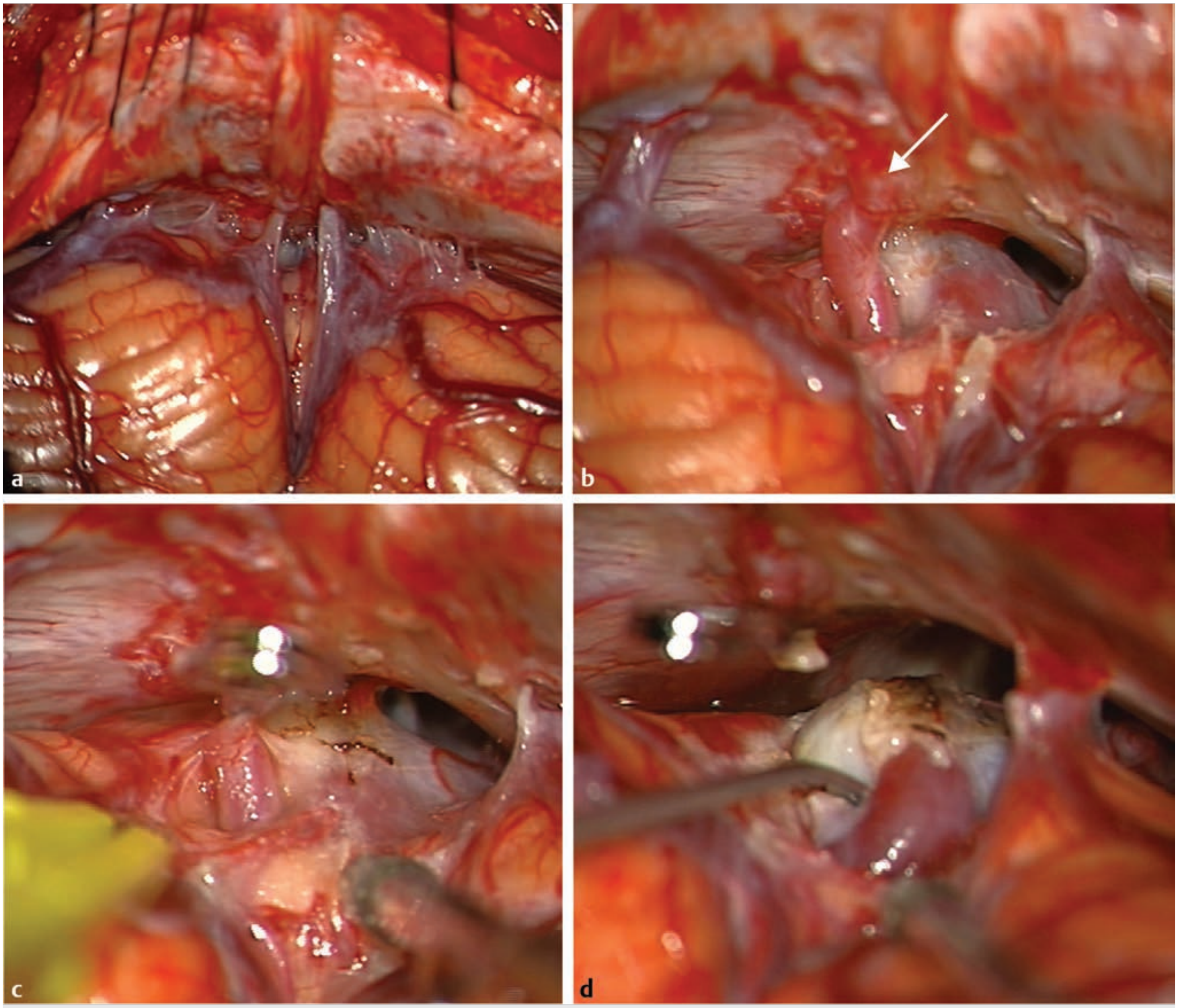

Fig. 33.5 Straight sinus dural arteriovenous fistulas (DAVFs) (type II). These DAVFs are supplied by the posterior meningeal artery and are drained by a vein coursing over the superior surface of the cerebellum, under the apex of the tentorium. **(a)** Intraoperative photograph demonstrates the supracerebellar-infratentorial approach, with the patient in the sitting position, torcular dura pulled superiorly with tacking sutures, and gravity retraction of the cerebellum to open the infratentorial plane. **(b)** The arterialized vein draining the fistula (*arrow*) is seen exiting the dura and coursing anteriorly to the galenic region. **(c)** The fistula was interrupted with a clip on the vein as it exited the tentorial dura, then **(d)** the vein was cut to obliterate the fistula.

venous outflow is occluded in a manner similar to that of type III fistulas, the interruption of the arterialized draining vein must preserve shunt flow into the torcular sinuses to preserve the major sinuses. Because less complete venous disruption is tolerated, arterial flow might also require disruption of the arterial inflow. In that case, the torcula must be skeletonized to identify and selectively ligate the arterial sources of the fistula.[24]

The arterial supply can feed into the torcula from all three spatial planes: above and below from the falx cerebri and cerebelli, from either side of the tentorium, and from any cortical dural surface in the occipital or suboccipital regions. Visualization of torcular skeletonization can be achieved using two incisions. Two incisions are required for each occipital cortical dural surface, which creates a corner at the transverse and superior sagittal sinuses. Similarly, a corner needs to be cut along the tentorium at the junction of the straight and transverse sinuses. A torcular craniotomy is adequate to visualize all eight of the dural leaflets, but retraction is required to make the tentorial cuts along the straight sinus.

33.3.4 Tentorial Sinus DAVFs

Tentorial sinus DAVFs are not well described, likely because the tentorial sinus itself is an obscure anatomical entity. The tentorial sinus can be found on magnetic resonance imaging (MRI) and diagnostic angiography, but it is often not thought to be of clinical or pathologic importance. The tentorial sinus can also vary widely in shape and location, and many practitioners may not group fistulas involving it into the same subtype. There have been multiple attempts by Rhoton and others to classify tentorial sinus anatomy.[25,26,27] Given the confusion in the literature over this subtle and highly variable anatomical structure, the relationship between the tentorial sinus and tentorial DAVFs off-midline has not been extensively described or firmly established.

DAVFs of the tentorial sinus are not associated with other sinuses within the dura. Some are located more laterally near the vein of Labbe where it tends to join the tentorial sinus under the occipital and temporal lobes, and others are medially located, where the medial variant of the tentorial sinus has been described.

Smith and Spetzler[28] previously outlined the supratentorial-infraoccipital approach for temporal lobe lesions near the posteromedial border with the occipital lobe, and in the author's experience, this approach is ideal for tentorial DAVFs. Although both lateral and prone patient positioning is acceptable for this approach, we favor the lateral positioning with the head rotated downward toward the floor. With the priority on a wide dural opening and the ability to freely mobilize the occipital lobes, particularly in medially located fistulas, a torcular craniotomy is indispensable. If the tentorial sinus fistula is more laterally located, for example, near the transverse-sigmoid junction, a midline opening may not be necessary. If this is the case, a unilateral craniotomy in the appropriate temporo-occipital region is adequate when taken down to, or below, the transverse sinus. For these fistulas to be occluded, the draining vein should be isolated and interrupted as it leaves the tentorial surface.

33.3.5 Superior Petrosal Sinus DAVFs

The extended retrosigmoid approach is an ideal exposure for superior petrosal sinus DAVFs, which have consistent infratentorial drainage into the petrosal vein (Dandy's vein).[29] Only the draining vein needs to be identified to disrupt the fistula, making more extensive transpetrosal approaches unnecessary. Creating an optimum view between the tentorium and the petrous face should be the aim of patient positioning. To achieve this goal, we prefer a lateral position with the head slightly flexed and angled downward in the direction of the floor. A curvilinear incision posterior to the ear is appropriate to perform a limited posterior mastoidectomy, skeletonization of the sigmoid sinus, a craniotomy (in favor of a craniectomy), and tacking of the dura to gently move the sigmoid sinus anteriorly. Although the typical retrosigmoid craniotomy does not involve drilling over the sigmoid sinus, the additional bony exposure in the extended retrosigmoid improves the viewing angles available to the surgeon relative to a traditional retrosigmoid working corridor.[29] Given the fistulous blood supply, drilling with a diamond drill bit and using a copious amount of bone wax are helpful, as the exposure process is bloodier while traversing the mastoid and petrous bones because of feeding arteries from the external carotid artery.

If the fistula has hemorrhaged and there is significant edema in the cerebellum, the first goal should be to release cerebrospinal fluid from the cisterna magna immediately after opening the dura in order to reduce cerebellar compression and improve exposure. Dissection using a standard microsurgical technique into the cerebellopontine angle leads to the arterialized petrosal vein, which is often variceal given the high-flow nature of this fistula (▶ Fig. 33.6). Vein avulsion is possible on clip application, and the clip should be placed close to the petrous dura without putting tension on the point where the vein directly meets the dura. Draining veins distal to the clip should be monitored to ensure transition to a bluish color after flow interruption to confirm that the fistula was occluded. Since venous varices can often hide an additional draining vein tracking in a medial direction toward the brainstem, the varix should be mobilized and this medial region should be inspected carefully. In our experience, approximately half of superior petrosal sinus DAVFs are Borden type II fistulas, with drainage medially into the superior petrosal sinus. It is possible for Borden type II fistulas to convert to low-risk type I fistulas after occlusion of the draining petrosal vein due to continued patency of the superior petrosal sinus.

33.3.6 Incisural DAVFs

Incisural DAVFs are uncommon and poorly characterized entities likely related to veins passing along the free edge of the tentorial incisura in which the basal vein of Rosenthal and lateral mesencephalic veins contribute to the fistula inflow.[13,30] This venous anatomy is not seen in most people. Incisural and tentorial sinus DAVFs are similar but distinct. Both are anatomically variable, are associated with the tentorial sinus, and almost always drain supratentorially. Despite these similarities, in our experience, they require different surgical approaches. As we described previously in the section on tentorial sinus DAVFs, tentorial sinus DAVFs are typically under the occipital lobe and are adequately exposed through a torcular or occipital craniotomy. In contrast, reaching an incisural DAVF surgically requires a more lateral subtemporal approach or a transsylvian approach through a pterional exposure. Venous outflow from incisural DAVFs usually courses in a posterior direction toward the vein of Galen or straight sinus; however, the fistulas often reside relatively anteriorly at the level of the mesial temporal lobe or near the intradural internal carotid artery. Incisural DAVFs are also in proximity to the superior petrosal sinus and can be confused with the more common petrosal DAVF. It is important to study the angiography and determine if a DAVF is petrosal or incisural because the former is easily reached with an extended retrosigmoid approach, whereas with the latter lesion, this approach would leave the surgeon approaching the tentorial edge from the inferior direction, preventing adequate access to the fistula even if the tentorium is split. Therefore, incisural DAVFs can be deceptive and necessitate close analysis of the venous outflow pattern on preoperative angiography to be certain of their type and location along the incisura. It is reasonable to suspect that some of the reported difficulty in surgically occluding superior petrosal sinus DAVFs is related to these subtle anatomical differences between superior petrosal sinus DAVFs and incisural DAVFs and to the inappropriate use of an infratentorial approach to incisural DAVFs.

33.4 Posterior Fossa

33.4.1 Transverse-Sigmoid Sinus DAVFs

Transverse-sigmoid sinus DAVFs are the second most common type in our surgical experience, comprising about 35% of those

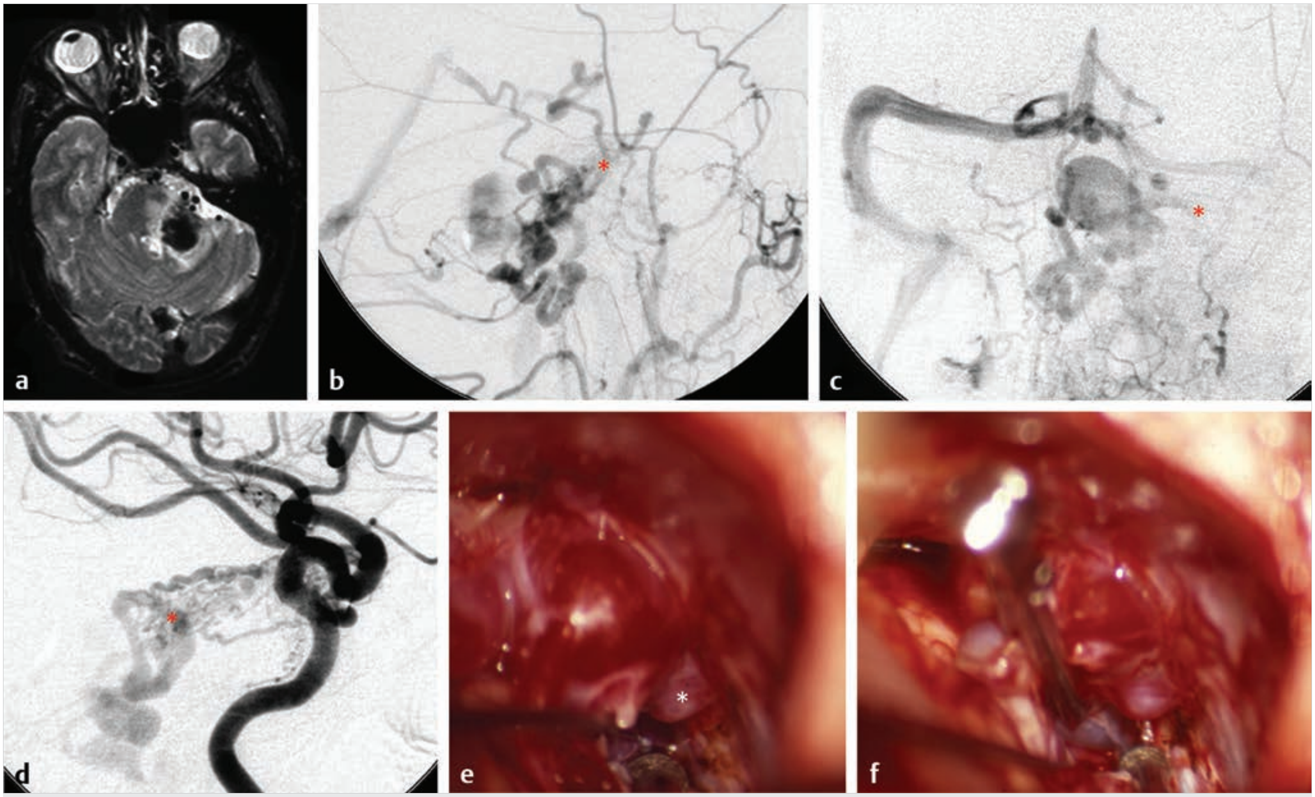

Fig. 33.6 Superior petrosal sinus dural arteriovenous fistulas (type V). **(a)** Brain magnetic resonance imaging (axial view, T2-weighted image) demonstrated a hematoma in the left cerebellar peduncle, with surrounding edema and dilated veins in the cerebellopontine angle. **(b)** Left external carotid artery angiogram (lateral view) showed an arterial supply to the fistula (*red asterisk*) from the middle meningeal artery and transosseous perforators. **(c)** The venous phase of the angiogram (anteroposterior view) showed the drainage of the fistula (*red asterisk*) through tortuous and variceal cerebellar veins. **(d)** Left internal carotid artery angiogram (lateral view) showed arterial supply to the fistula (*red asterisk*) from the tentorial artery. **(e)** Intraoperative photograph demonstrates the extended retrosigmoid approach, with the patient in the lateral position (right side down), the dura flapped against the transverse and sigmoid sinuses, and exposure of the angle between the petrous bone and the tentorial dura. The vein draining the fistula was visualized at the petrotentorial junction (*white asterisk*). **(f)** The fistula was interrupted by placing a clip on the draining vein. (Reproduced with permission from Lawton MT, Sanchez-Mejia RO, Pham D, et al. Tentorial dural arteriovenous fistulae: operative strategies and microsurgical results for six types. Neurosurgery 2008;62:110–124, discussion 124–125.)

surgically treated. These lesions are readily treatable with endovascular approaches. Transvenous and transarterial approaches can be used alone or together to occlude these types of fistulas. Given the high cure rates with endovascular therapy, these lesions rarely require surgical intervention. If surgical treatment is pursued, these lesions can be approached with an extended retrosigmoid craniotomy that adds width to the junction of the transverse and sigmoid sinuses by drilling off additional overlying bone with a diamond drill bit, as described previously for superior petrosal sinus DAVFs. After the dura is lifted, the arterialized draining vein should be located and divided. It is also prudent to skeletonize the transverse, sigmoid, and superior petrosal sinuses for Borden type II fistulas. If angiography demonstrates a Borden type I fistula, it may be managed conservatively.

33.4.2 Marginal Sinus DAVFs

The marginal sinus is a circumferential structure that sits at the level of the foramen magnum, and fistulas involving it are rare. It encircles the cervicomedullary junction and has venous outflow into the jugular veins or sigmoid sinuses. These DAVFs are typically supplied by branches from the vertebral artery, including the posterior meningeal artery and directly from small branches of the vertebral artery trunk. Small muscular branches can also penetrate to provide arterial flow, including those from the external carotid circulation. Aside from hemorrhage, patients can present with signs of venous hypertension on MRI, or with symptomatic manifestations due to brainstem or cervical cord compression from variceal veins.

Marginal sinus DAVFs usually are readily treated by endovascular occlusion, resulting in a high cure rate. They rarely require surgical intervention. Given the low-lying position of these lesions near the cervicomedullary junction, the ideal tactic is the far-lateral approach. Opening the dura often occludes feeders arising from the posterior meningeal artery. In addition, the far-lateral approach allows quick access to feeding vessels arising from the subarachnoid segment of the vertebral artery. The arterialized vein is then clipped, cauterized, and divided as it leaves the dura covering the occipital condyle.

33.4.3 Inferior Petrosal Sinus DAVFs

Along with marginal sinus DAVFs, inferior petrosal sinus DAVFs are very rarely encountered. They usually do not have leptomeningeal venous drainage and often follow a benign clinical course. Patients with inferior petrosal sinus DAVFs rarely present with hemorrhage, and most of these lesions are found incidentally or when the patient presents with pain or cranial nerve deficits. If these lesions are symptomatic and do not improve with conservative management, endovascular therapy should be the first treatment option considered.

33.5 Decision-making and Preoperative Preparation

The decision to aggressively treat DAVFs is usually made solely on the basis of the severity of neurologic symptoms and angiographic characteristics, and DAVFs in the posterior fossa should be uncompromisingly treated to prevent hemorrhage.[7] Catheter angiography is typically used to separate DAVFs into risk groups based on the venous drainage pattern and should consist of a six-vessel study. Although angiography provides exceptional anatomical resolution of the fistula site, blood supply, and drainage, it offers a rough estimation of cerebral perfusion. Conventional MRI and magnetic resonance angiography may show hypertrophied dural feeders, distended cortical veins, and parenchymal signal changes indicative of venous ischemia. Perfusion computed tomography may be the best and simplest way to assess any abnormal hemodynamics in patients with DAVFs. With cerebral perfusion data, venous hypertension and ischemia may be assessed, which may provide a way to individualize patient treatment decisions beyond the assessment of hemorrhagic risk.

In all but the most minor fistulas, preparations should be made for major blood loss that can occur from skin incision to craniotomy, to removing the bone and dealing with the fistula.

33.6 Postoperative Management and Possible Complications

Catheter angiography is recommended after surgery to ensure that the cortical venous reflux has been completely eliminated.[18] If residual cortical venous reflux is found, then further treatment with surgery, endovascular embolization, or stereotactic radiosurgery is indicated.[31] Long-term neuroimaging surveillance is often prudent since there is some risk of recurrence. Complications specific to surgery include brain swelling from unplanned venous occlusions and postoperative hemorrhage.

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34 Anterior Fossa, Superior Sagittal Sinus, and Convexity Dural Arteriovenous Malformations

Brian T. Jankowitz, Paul A. Gardner, Michael McDowell, Xiao Zhu, and Robert M. Friedlander

Abstract

Dural arteriovenous fistulas (DAVFs) are abnormal arteriovenous shunts within the dura. About 10 to 15% of all intracranial arteriovenous malformations are DAVFs. DAVFs of the anterior fossa and superior sagittal sinus represent about 6 and 7% of reported DAVFs, respectively. They are often associated with retrograde leptomeningeal venous drainage and by virtue of this, an aggressive clinical course mandating treatment.

Keywords: dural arteriovenous fistula, superior sagittal sinus, anterior cranial fossa, microsurgery, leptomeningeal venous drainage

34.1 Patient Selection

34.1.1 Indications

Patients typically present with symptoms due to hemorrhage, ischemia, or hydrocephalus. They may also present with headache or a bruit, although bruits are less common than with transverse and sigmoid sinus dural arteriovenous fistulas (DAVFs). The overall risk of hemorrhage from a DAVF is 2% per year, but hemorrhage is rare unless there is leptomeningeal venous drainage, in which case the risk rises to 8% per year. Some studies suggest that DAVFs with leptomeningeal venous drainage have a risk of hemorrhage of 2% per year unless they have a history of hemorrhage, in which case the risk of hemorrhage increases to 7% per year.[1,2] The hemorrhage may be intraparenchymal, subarachnoid, subdural, and/or intraventricular and may be distant from the site of the dural nidus. Symptoms of hemorrhage include headache, seizures, focal deficit, or decreased level of consciousness.

Neurological symptoms not related to hemorrhage can result from venous congestion. Congestive ischemia may cause a focal deficit or seizures. Hydrocephalus may result from venous sinus hypertension or progressive arachnoid villous fibrosis due to repeated subarachnoid hemorrhage. Chronic neurological deficits may improve with treatment of an underlying DAVF.

The principal indications for treatment of a DAVF include prior hemorrhage, neurological deficit, or high risk of hemorrhage based on angiographic features. Expeditious treatment is recommended for any DAVF that has hemorrhaged and generally for those with leptomeningeal venous drainage.[2] Patients without these indications may be observed, with intervention considered if symptoms such as headache or bruit are unbearable.

34.1.2 Imaging and Anatomical Considerations

The most useful imaging modality is catheter angiography, which should include selective injections of the internal, external, and vertebral arteries (▶ Fig. 34.1, ▶ Fig. 34.2, ▶ Fig. 34.3). Useful information can be gathered from computed tomography (CT) and magnetic resonance imaging (MRI) regarding anatomical location as well, but catheter angiography remains the gold standard. CT remains the most practical and sensitive method for detecting hemorrhages and may also show hydrocephalus or prominent venous channels in the calvarium. MRI is most useful for detecting ischemia, edema, or hemosiderin deposition from old hemorrhage. CT and MR angiography and venography may show an occluded sinus as well as anatomical details such as dilated cortical veins (▶ Fig. 34.4). Image guidance may be helpful to localize convexity and sagittal sinus lesions.

A useful way to classify DAVFs is the Borden system.[3] Borden type I DAVFs do not have venous sinus obstruction, and

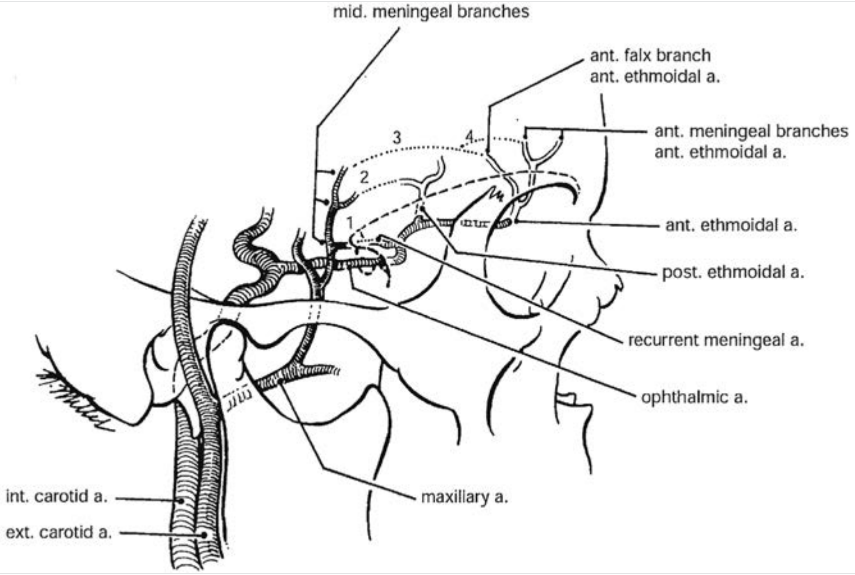

Fig. 34.1 Normal blood supply to the anterior fossa dura. The four sites of anastomosis between the ophthalmic artery and meningeal branches of the external carotid artery are shown. (1) Middle meningeal artery to the recurrent meningeal artery. (2) Middle meningeal artery to the meningeal branch of the posterior ethmoidal artery. (3) Middle meningeal artery to the anterior falcine branch of the anterior ethmoidal artery. (4) Middle meningeal artery to the meningeal branch of anterior ethmoidal artery. (Reproduced with permission from Patel AB, King WA, Martin NA. Operative management of anterior fossa, superior sagittal sinus and convexity dural arteriovenous malformations. Neurosurgical Operative Atlas. 1st ed. 1999;8:70.)

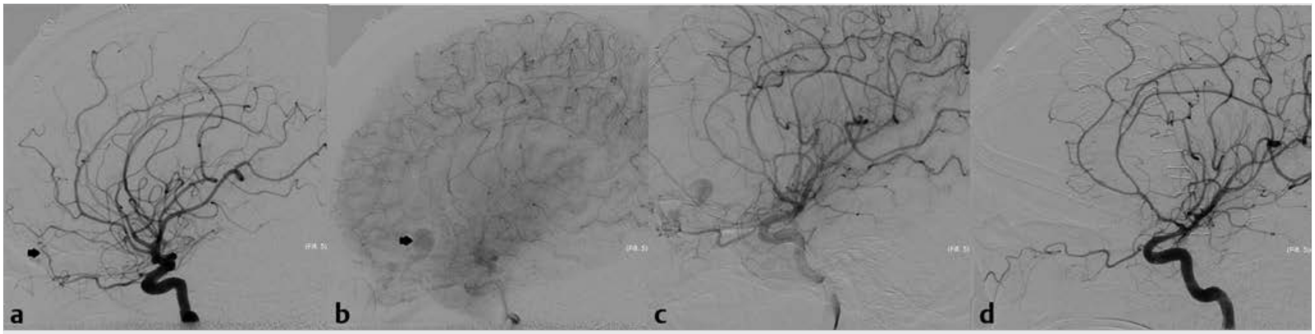

Fig. 34.2 Diagnostic angiogram demonstrating a superior sagittal sinus fistula. **(a)** The fistula was supplied exclusively by the anterior ethmoidal artery in the early arterial phase (arrow), **(b)** there was venous varix noted during the late venous phase, **(c)** both feeding and early venous drainage are seen clearly in the late arterial phase preoperatively), **(d)** complete absence of flow is noted in the later arterial phase postoperatively.

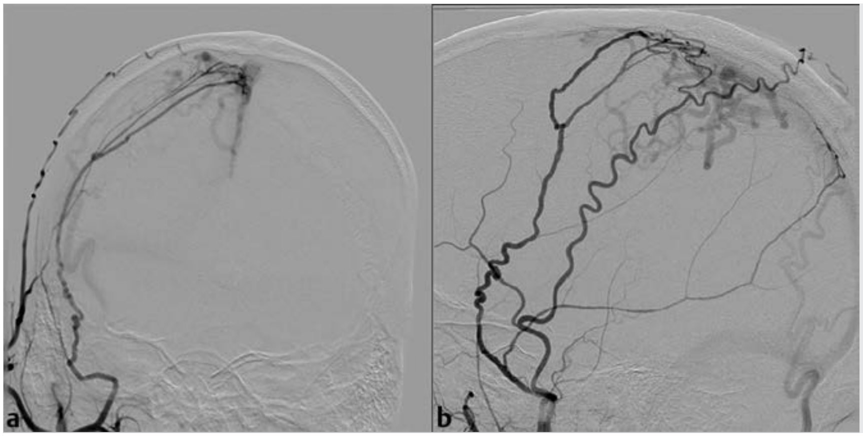

Fig. 34.3 Diagnostic angiogram demonstrating a superior sagittal sinus fistula. **(a)** Anteroposterior view demonstrating dual supply by both the middle meningeal artery and the superficial temporal artery, **(b)** lateral view demonstrating dual supply by both the middle meningeal artery and the superficial temporal artery.

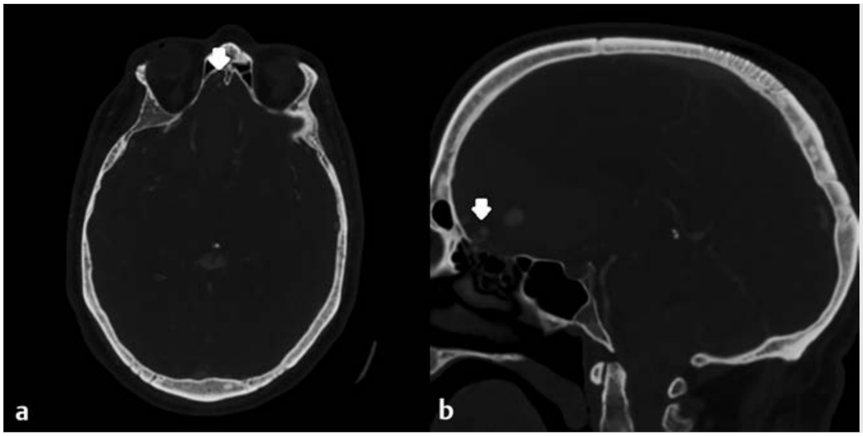

Fig. 34.4 Computed tomography angiogram demonstrating the anatomic location of a superior sagittal sinus fistula. **(a)** Axial view demonstrating the arterial ethmoidal feeding vessel, **(b)** sagittal view demonstrating the arterial feeder as well as associated venous varix deep to the feeder.

venous drainage is anterograde through a patent venous sinus and meningeal veins with no retrograde leptomeningeal drainage. Type II DAVFs have anterograde venous sinus and meningeal flow with retrograde leptomeningeal venous drainage. Type III DAVFs drain only via retrograde leptomeningeal venous flow. Classically, only types II and III are considered at risk for hemorrhage. DAVFs of the anterior fossa are more likely than those of the sigmoid or transverse sinus to be types II and III, accounting for their increased risk of hemorrhage.

Arterial supply is typically from the anterior ethmoidal artery and is bilateral in about half of the cases. Additional feeders may arise from the posterior ethmoidal artery, anterior falx artery, and branches from the external carotid system. Venous drainage usually occurs via cortical veins that drain to the superior sagittal and/or cavernous sinuses.

Superior sagittal sinus DAVFs typically involve the middle and posterior thirds of the sinus. They may be subclassified into those that drain directly into the superior sagittal sinus and are at a very low risk of hemorrhage and those that drain via a cortical vein and are at high risk of hemorrhage. Arterial supply is typically from the middle meningeal artery and may be bilateral with additional supply from the superficial temporal and occipital arteries (▶ Fig. 34.3).

34.1.3 Treatment Options

These include observation, compression therapy, endovascular therapy, open surgery, and stereotactic radiosurgery.[4,5,6] Almost all DAVFs without leptomeningeal drainage can be managed conservatively; however, most anterior fossa or superior sagittal sinus DAVFs exhibit leptomeningeal drainage. With low-risk DAVFs, manual compression of the principal arterial supply may lead to resolution of the fistula in approximately 30% of the cases. Compression therapy for anterior fossa DAVFs is not recommended due to concerns of ischemic stroke, hypotension, and bradycardia. With superior sagittal sinus DAVFs fed only by branches of the superficial temporal artery, compression therapy may be attempted.

Endovascular treatment of DAVFs requires placement of embolic material at the site of the fistula and preferentially extending into the draining vein.[5] Coils have been replaced by liquid embolics as the mainstay of endovascular treatment. This can be accomplished via a transarterial or transvenous route from the internal carotid artery, external carotid artery, or internal jugular vein. In extreme circumstances, if there is an arterialized segment of superior sagittal sinus, a burr hole can be drilled over the sinus, which is approached with an angiographic catheter for coil-occlusion of the sinus. However, it is important not to disrupt normal venous drainage, so this approach should not be used when there is anterograde flow through the sinus. When surgery is considered too great a risk and embolization is unlikely to cure the fistula alone, partial embolization may be appropriate for palliation of symptoms or as an adjunct to radiosurgery.

Radiosurgery has been used to treat DAVFs, with complete obliteration documented in up to two thirds of patients in initial studies.[6] Patients with small, localized fistulas are the best candidates, and this strategy has also been used for patients who are poor candidates for open microsurgical or endovascular treatment. Adjunctive embolization after radiosurgery may be used for palliation of symptoms and possibly to decrease the risk of hemorrhage.

DAVFs often have anatomic considerations that make them more amenable to an open or an endonasal approach. The floor of the anterior cranial fossa and the superior sagittal sinus, where arterial access is impaired by vessel size and distance or where sacrifice of delicate venous drainage is undesirable. If preoperative angiography demonstrates feeding vessels with significant contribution to eloquent structures, these are often better managed selectively with surgery or a combined approach. Further, pediatric patients presenting with DAVFs are more likely to require a surgical or multimodality approach. Endovascular therapy alone carries greater risk in children when compared to adults due to difficult with selective microcatherization, femoral access, and limits in contrast quantity as well as ionizing radiation tolerance.

34.2 Preoperative Preparation

Precautions for possible air embolism, including a central venous catheter, precordial Doppler, and endtidal CO_2 monitoring are used. Perioperative coverage with antiseizure medicines is recommended, as is standard antibiotic prophylaxis. Furosemide and mannitol are administered to achieve appropriate brain relaxation. A radiolucent head holder should be placed to facilitate the use of intraoperative angiography. Ability to intraoperatively manipulate the positioning of the head, in particular the height of the head with respect to the heart, is highly desirable. This can assist in both regulation of venous bleeding and enhancement of the operative corridor. With any sizeable DAVF there is a risk of torrential hemorrhage for which provisions should be made including availability of blood and good venous access.

34.3 Operative Procedure

34.3.1 Anterior Fossa Dural Arteriovenous Fistulas

The patient is positioned supine with the head elevated to maximize venous drainage and extended to minimize retraction of the frontal lobes. A lumbar drain may be placed to facilitate exposure. A unilateral subfrontal approach on the side of the DAVF is preferred to avoid bilateral olfactory nerve injury (▶ Fig. 34.5). If contralateral leptomeningeal draining veins are present, these may be treated via a transfalcine approach. Rarely, extensive involvement of the falx or dura along the floor of the anterior fossa will require a bifrontal approach. In rare cases, a transfrontal sinus approach may allow for better surgical precision, such as in cases where the fistula is closely adherent to midline and anterior or in situations where there is substantial concern for significant dural

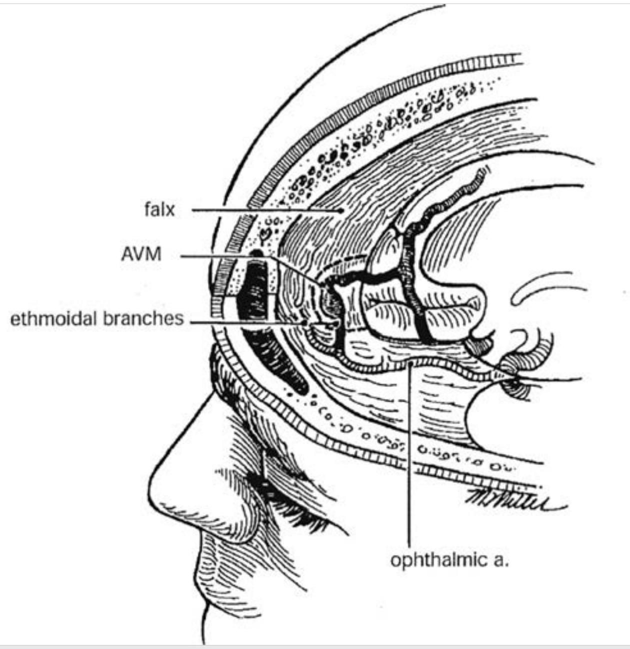

Fig. 34.5 Anterior fossa dural arteriovenous fistula in the region of the cribriform plate and the anterior falx. The lesion has an enlarged anterior ethmoidal artery with a fistulous connection to the leptomeningeal veins. This connection is dilated into a varix, which is usually the site of hemorrhage. The operative goal is to obliterate this fistulous connection. (Reproduced with permission from Patel, King, Martin. Operative management of anterior fossa, superior sagittal sinus and convexity dural arteriovenous malformations. Neurosurgical Operative Atlas. 1st ed. 1999;8:71.)

adhesion to the inner table. The frontal sinus adjacent to the bone may be drilled with less risk to the dura and minimally exposes brain parenchyma while allowing visualization of select DAVFs without retraction. This approach may require deepithelialization of the frontal sinus depending on the extent of exposure and potentially could increase risks of infection. A robust sinus must also be present.

The above craniotomies may be created through a bicoronal skin incision behind the hairline. The incision must start above the level of the zygomatic arch and less than 1 cm anterior to the tragus to avoid injury to the frontal branch of the facial nerve. The skin is elevated superficial to the temporalis fascia. Pericranium should be elevated with skin and protected for potential dural grafting. Three burr holes are placed for elevation of the bone flap. The first is placed at the keyhole. To do this the temporalis muscle will need to be elevated just from that area. Two burr holes are then placed along the midline just lateral to the superior sagittal sinus (ipsilateral to the craniotomy), one just above the orbital rim and the other one 8 to 10 cm superior to this. The exact location of the anterior frontal burr hole depends on the precise location of the fistula, with a low burr hole required for subfrontal exposure, which is the usual case. The dura under the planned bone flap is stripped carefully using a dental instrument and a Penfield 3 and the bone flap is created using a craniotome.

Given that the venous system may have different degrees of arterial pressure, the surgeon must be prepared for potential rapid blood loss during the elevation of the bone flap. Two common sites of blood loss can be the dura or the edge of the superior sagittal sinus or the bone itself. DAVFs may form large draining channels in the bone proper, resulting in significant bleeding upon elevation of the bone flap. Bleeding can be controlled with bone wax and elevation of the head. During the craniotomy, the frontal sinuses may be entered. To minimize brain retraction, this should not be avoided in patients with large frontal sinuses. If the sinus is entered, the mucosa is stripped, the ostium of the sinus is plugged with a small piece of temporalis muscle and the cavity covered with a flap of pericranium. To facilitate exposure and decrease brain retraction, the lateral aspect of the sphenoid wing may be removed by drilling. Next the dura may be tacked to the bone edge using 4–0 braided nylon sutures if there is epidural bleeding. If not, we place the dural tackups after dural closure. This permits dural relaxation, simplifying the closure. Attention again should be paid to the possibility of entering the frontal sinus during placement of inferior and anteromedial tackup sutures.

The dura is opened, allowing three flaps to be reflected medially toward the superior sagittal sinus, inferior over the orbital rim and laterally. If a cerebral hematoma is present, it is carefully evacuated to improve visualization and minimize brain retraction. Fragile, dilated draining veins will be present in or next to the clot and these must be avoided. The frontal lobe is covered with moist Telfa (Tyco Healthcare/Kendall, Mansfield, MA) or Gelfoam (Pfizer Inc., New York, NY) and cottonoids and is gently retracted. Care must be taken not to avulse the draining veins that are attached to the dura near the cribriform plate. The DAVF is recognized most often as single but also can be multiple connections from the dura to pial veins. All of these fistulas must be cauterized and divided. This is the critical part of the procedure. Dural resection and excision of venous structures are avoided because these risk cerebrospinal fluid leakage and parenchymal injury, respectively, without benefit. Indocyanine green videoangiography may be helpful to evaluate for early venous drainage and distinguish it from normal venous outflow. Ultrasonography may also be utilized to identify draining veins that feed into the fistula. Typically, drainage into the fistula is high velocity and highly pulsatile, similar to arterial signal. If there is concern for residual flow, Doppler ultrasonography may also assist in confirmation. Arterial supply to the fistula that may be present in dura or other opaque tissues may also be traced. Intraoperative catheter angiography can also be helpful to ensure complete eradication of the fistula, but the added time, awkward positioning, and need for selective angiography diminishes its utility.

The dura is closed in a watertight fashion and the bone flap is attached using titanium plate fixation, taking care to avoid any gap in the bone on the patient's forehead. We routinely use methylmethacrylate to achieve a cosmetic result. The scalp is then closed in layers after the placement of a subgaleal 7 French Jackson Pratt drain when needed.

In some circumstances, anterior DAVFs involving supply from ophthalmic artery branches may be targeted with an endoscopic endonasal approach in collaboration with otolaryngology (▶ Fig. 34.6**, Video 34.1**). If such a technique is pursued, the patient in placed in the supine position in Mayfield head-pin fixation with image guidance. Oxymetazoline packing is inserted into the nasal passages and allowed to decongest the nasal passages while the patient is prepped and draped. The pledgets are then removed and an initial inspection of the nasal passages is performed with a zero degree endoscope. A vascularized nasal septal flap should be elevated and placed into the nasopharynx for reconstruction after the procedure. The vascular pedicle from the posterior nasal artery must be preserved carefully. The anterior tip of the right middle turbinate can be resected and the mucosa medial to it and anterior to the cribriform plate cauterized. The superior septum is then resected to provide binarial exposure. The nasofrontal recess is exposed on both sides and all intervening soft tissue is removed. The

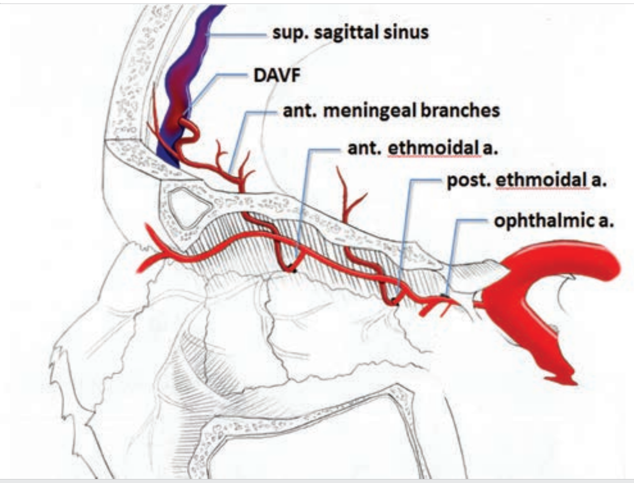

Fig. 34.6 Diagram of a superior sagittal sinus fistula drained by feeders of the ophthalmic artery that was treated via endoscopic endonasal approach for obliteration. Relevant branches of the ophthalmic artery are displayed.

anterior ethmoid air cells are opened and the anterior cranial base visualized. The anterior ethmoid arteries are cauterized if angiographic or visual evidence suggests involvement with the DAVF. All remaining mucosa is removed from the anterior cranial base in the area of the crista galli. The cranial bone is drilled and removed to expose the overlying dura, which is cauterized and incised to visualize the DAVF and its supplying arteries. Feeding arteries should individually be inspected and cauterized. Once all visible vessels are eliminated, the falx may be dissected and cauterized to eliminate any further vessels feeding the DAVF superiorly. The margins of the DAVF may then be inspected and the remaining specimen resected once coagulation of the draining vessel has been performed. A multilayer closure including the previously prepared nasoseptal flap may be used to seal the defect created from the procedure once the field has been carefully irrigated and hemostasis achieved (**Video 34.1 and** ▶ Fig. 34.7).

34.3.2 Superior Sagittal Sinus and Convexity Dural Arteriovenous Fistulas

The patient is positioned so that the DAVF will be the highest point in the operative field (▶ Fig. 34.8). Image guidance is of assistance to optimize positioning as well as surgical approach. For lesions in the anterior one third of the superior sagittal sinus, the patient is positioned supine with a shoulder roll on the side of the lesion. The head is fixed in neutral position. A bicoronal skin incision is used. If the lesion is in the middle third of the superior sagittal sinus, the patient is positioned supine with the head flexed to access the lesion, but without tension in the neck and with at least two fingerbreadths between the chin and the chest. Again a shoulder roll is used on the side of the

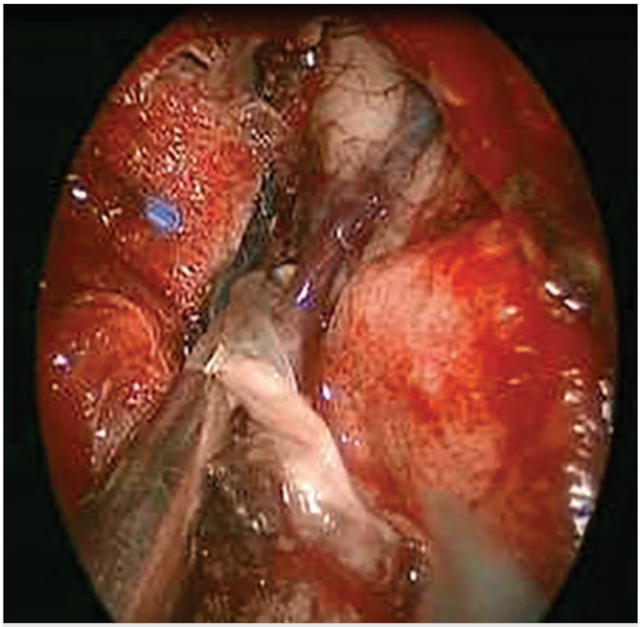

Fig. 34.7 Endoscopic endonasal view of a superior sagittal sinus arteriovenous fistula with venous component and arterial feeder visualized.

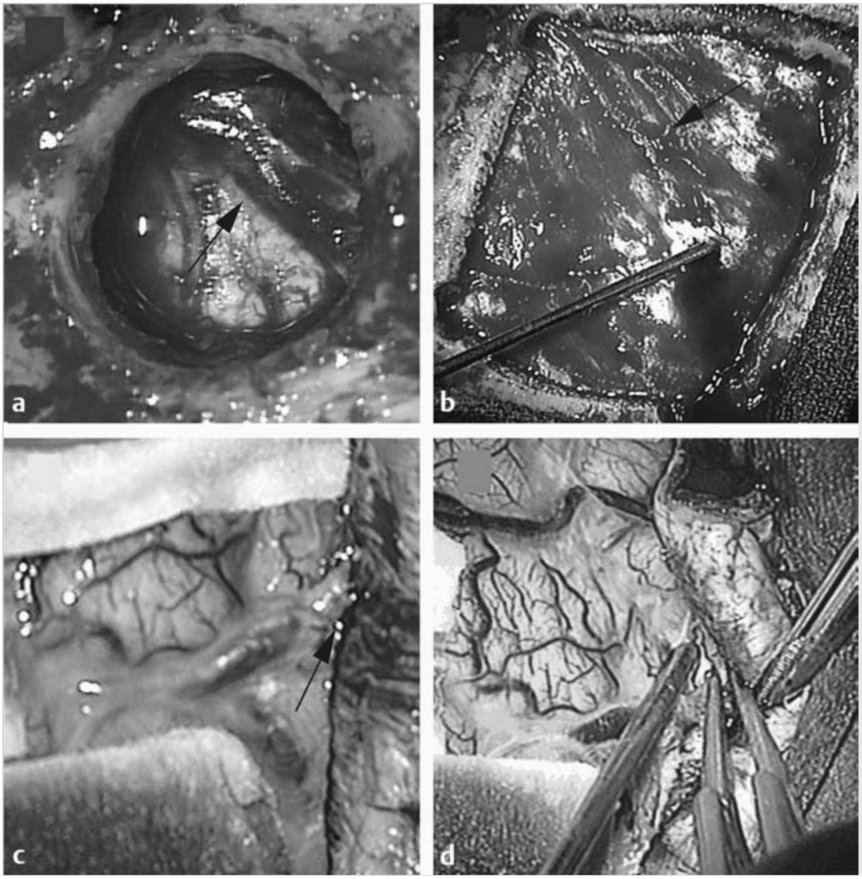

Fig. 34.8 A convexity dural arteriovenous fistula (DAVF) showing intraoperative photographs where anterior is toward the top of the photograph and midline is to the right of the photograph. **(a)** Initial anterolateral burr hole showing a middle meningeal arterial feeder *(black arrow)* to the DAVF. **(b)** Middle meningeal artery feeder *(black arrow)* visualized after turning the bone flap. **(c)** Upon opening dura, the fistulous connection *(black arrow)* was observed between the cortical vein and the dura. **(d)** After dissecting the vein from the arachnoid, the fistula was obliterated with bipolar coagulation and then divided.

lesion. A U-shaped incision is made. For lesions in the posterior one third of the superior sagittal sinus, the patient is positioned prone and a U-shaped incision is used. Alternatively, a midline or parasagittal linear incision may be used, which decreases the risk of injury to the greater occipital nerve.

The skin incision and bone flap are fashioned to allow exposure to the lesion and both sides of the superior sagittal sinus. This is usually done with four burr holes placed at the corners of the bone flap. The dura over the superior sagittal sinus can be stripped from the bone with a dental instrument and Penfield 3. If the dura is adherent, which is more common in older patients, the initial bone flap may be removed up to the edge of the superior sagittal sinus. This can be stripped under direct vision and then an additional bone flap extending across the superior sagittal sinus is removed. Substantial bleeding can occur during the craniotomy due to large arterialized draining venous channels in the bone. The bone windows of the CT should be inspected preoperatively to determine whether this is the case. After removing bone, a sinus pattie is placed over the superior sagittal sinus for hemostasis. Additional hemostasis is obtained with bipolar coagulation. Again, substantial and even catastrophic dural bleeding can occur, particularly over the fistula. This is best controlled by covering it with Gelfoam and applying pressure with a pattie. Next the dura is tacked to the bone as needed, as described earlier.

Great care is required upon opening the dura. A U-shaped dural opening centered upon the malformation and flapped toward the midline will provide ideal exposure. However, cortical veins are commonly attached to the inner surface of the dura. These should not be separated from the dura. The dural flap should be fashioned around these critical veins. Once the dura is opened, retraction of the dura medially and the hemisphere laterally will assist in obtaining an ideal surgical corridor. However, too much retraction is avoided because it could compromise normal drainage of the superior sagittal sinus and bridging veins.

For lesions that drain via cortical veins, these draining veins should be coagulated as they exit the dura. It is possible that a leptomeningeal vein with retrograde flow will drain back into another part of the sinus. These are also coagulated. Occasionally, the fistula will be located in the wall of the superior sagittal sinus but will drain only via cortical veins. Again, these cortical veins are coagulated. If the DAVF drains only into the superior sagittal sinus, this fistulous connection and the wall of the superior sagittal sinus are coagulated, relieving venous hypertension in the sinus. In cases with an occluded sinus and collateral venous drainage, the involved portion of the superior sagittal sinus may be packed and excised. However, this is usually not necessary and the superior sagittal sinus cannot be occluded posterior to the coronal suture if there is anterograde flow. If the fistula is bilateral, the contralateral side should also be explored to make sure that there are no fistulous components. Intraoperative angiography may be used to confirm obliteration of the lesion.

The dura is closed primarily or with a pericranial graft or allograft. The bone flaps are reattached with titanium plates and screws and the scalp is closed in layers.

34.4 Postoperative Management Including Possible Complications

The patient should be monitored closely overnight, with systolic blood pressure controlled so as to prevent extreme elevations. A postoperative angiogram should be obtained before discharge. Antiseizure medicines beyond the immediate postoperative period are unnecessary, unless the patient has had seizures, in which case prolonged administration may be necessary.

Specific complications are neurological deficits due to venous ischemia or infarction, postoperative intracranial hemorrhage and residual or recurrent DAVF. There is no specific treatment for venous complications. Avoiding corticosteroids because of their prothrombotic activity may be warranted in general. Residual DAVF is generally best managed by immediate reoperation. Delayed recurrence can also occur, although it is still uncommon and whether to follow these patients for years with serial imaging is uncertain, but it is an option.

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Part III

Ischemic and Other Cerebrovascular Diseases

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III

35 Carotid Endarterectomy

Daphne D. Li, Paul D. Ackerman, and Christopher M. Loftus

Abstract
Several randomized clinical trials found that carotid endarterectomy (CEA) is superior to medical management alone for symptomatic patients with greater than 50% carotid stenosis and for asymptomatic patients with greater than 60% carotid stenosis. Percutaneous angioplasty and stenting is another option that is controversial but is used by some physicians to treat carotid stenosis, especially patients with anatomically difficult lesions, significant comorbidities, or other disease processes that render the anesthetic risk unacceptably high. In our experience, most patients with critical cervical carotid stenosis are well-served by open surgical carotid artery reconstruction. This chapter addresses the indications for CEA, including appropriate patient selection, optimal medical therapy, preoperative evaluation, complication avoidance, perioperative management, and appropriate surgical follow-up. Finally, our surgical technique is described in detail.

Keywords: carotid endarterectomy, external carotid artery, carotid bifurcation, asymptomatic carotid stenosis, symptomatic carotid stenosis, carotid occlusion, ischemic stroke, atherosclerosis, carotid stent

35.1 Patient Selection

35.1.1 Carotid Stenosis

The North American Symptomatic Carotid Endarterectomy Trial and the European Carotid Surgery Trial found that carotid endarterectomy (CEA) significantly decreased the incidence of ipsilateral stroke among patients with symptomatic carotid stenosis with greater than 70% carotid stenosis compared to medical management.[1,2] In addition, the latter study found that for a subset of high-risk patients, there was benefit of CEA for patients with high–moderate (50–69%) carotid stenosis. Several randomized clinical trials have also found that CEA reduces the risk of ipsilateral stroke in asymptomatic patients with greater than 60% stenosis.[3,4] For symptomatic patients, the benefit from CEA is greatest when it is performed within 2 weeks of the transient ischemic attack (TIA) or minor stroke.[5]

35.1.2 Carotid Occlusion

The efficacy of CEA in patients with carotid occlusion or near-occlusion is controversial and metaanalysis of data from randomized clinical trials concluded that evidence to support CEA in this setting was lacking but the number of patients studied was very limited.[6] Nevertheless, it is our practice to operate in the setting of subacute and chronic carotid occlusion, particularly if there is any suggestion of a "string sign" on carotid angiography that may be consistent with carotid patency and the patient is symptomatic and not disabled (▶ Fig. 35.1).

35.1.3 Carotid Stenting

Carotid angioplasty and stenting (CAS) with or without distal protection devices has been suggested as an alternative to CEA.[7,8,9] The decision to treat carotid stenosis by CEA or CAS is controversial. In general, randomized clinical trials comparing these procedures have shown that both reduce the risk of stroke, although it is not clear if non-inferiority of CAS compared to CEA has been demonstrated.[9] CAS is associated with a higher risk of periprocedural stroke whereas CEA is associated with a higher risk of periprocedural myocardial infarction. Restenosis may be more common after CAS.

In general, the most widely accepted indications for CAS are patients with multiple medical illnesses, high anesthetic risk, restenosis after prior CEA, radiation-induced stenosis, and anatomical variations principally a very high carotid bifurcation.

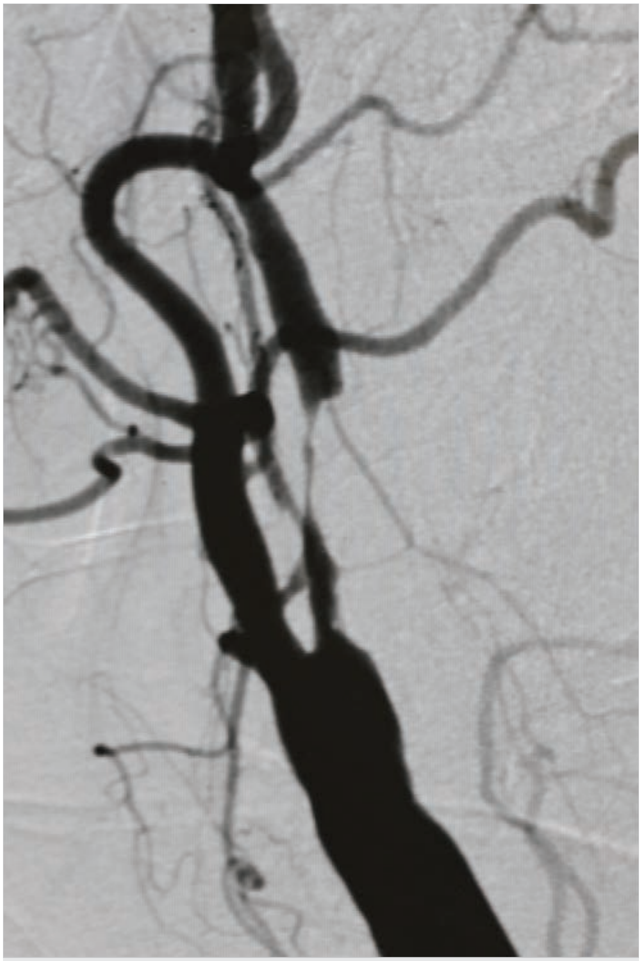

Fig. 35.1 Anterior–posterior projection of a common carotid injection demonstrating near-complete vessel occlusion and angiographic "string sign" along the proximal internal carotid artery just distal to the carotid bifurcation.

We remain unconvinced, particularly in regards to the elderly patient population, that CAS provides any tangible benefit to patients with carotid stenosis. In our series of patients older than 70 years, we found no difference in surgical outcome and further posit that the more friable and tortuous vessels commonly encountered in older patients only serve to complicate endovascular navigation.[10]

35.2 Preoperative Evaluation

35.2.1 Imaging Studies

There is no consensus regarding the appropriate preoperative vascular imaging required prior to CEA. At our institution, the diagnosis of carotid stenosis must be confirmed either by carotid duplex ultrasound or computed tomography arteriogram (CTA). CT or catheter angiography are required before surgery. CTA is usually sufficient for surgical planning. If there is any ambiguity about the vascular anatomy, or if carotid bulb calcification renders the CTA insensitive, then we obtain a catheter angiogram—which remains the gold standard for preoperative evaluation of carotid stenosis.

35.2.2 Medical Management

Anticoagulation and Antiplatelet Therapy

Meta-analyses of several randomized controlled trials have demonstrated that antiplatelet therapy with aspirin reduces the risk of stroke of any cause in patients undergoing CEA.[11] Patients with carotid stenosis may also be prescribed clopidogrel or warfarin for various indications; however, there is no direct evidence that either of those medications reduces the incidence of thromboembolic stroke related to carotid stenosis. Statins (HMG-CoA reductase inhibitors) may reduce carotid plaque progression and the incidence of transition from asymptomatic to symptomatic carotid stenosis. Other studies have also reported a clinically significant decrease in perioperative morbidity and mortality when patients are treated with a statin prior to CEA.[12] Therefore, it is our practice to recommend that all patients with carotid stenosis take daily aspirin, a recommendation based on robust high-quality data, and a statin, based on more limited lower quality evidence, unless there are pharmacologic contraindications.[13]

35.3 Operative Procedure

35.3.1 Positioning

The patient is positioned supine; the head cradled in a foam donut cushion and five to six towels are placed between the patient's shoulder blades to induce gentle cervical extension. The head is then turned approximately 15 to 30 degrees—depending on the relationship between the external carotid artery (ECA) and the internal carotid artery (ICA)—contralateral to the surgical side in order to maximize the distance between the jaw and the clavicle. Greater head rotation is required when the ICA is medially rotated (i.e., "hidden," or tucked underneath the ECA).

Two anatomical landmarks are identified on the preoperative radiographic studies to estimate the rostral extent of the required exposure. The first is the angle of the mandible, which is palpated and marked before skin incision. The second is the position of the carotid bifurcation, particularly its relationship with the distal extent of the cervical plaque.

35.3.2 Anterior Cervical Dissection

A longitudinal incision is marked parallel to the anterior margin of the sternocleidomastoid muscle and is extended according to anatomic landmarks discussed previously—sometimes as low as the sternal notch and other times as high as the posterior auricular area (▶ Fig. 35.2). The skin is infiltrated with local anesthetic prior to incision with a number 15-blade scalpel. The platysma is opened sharply, also in the rostral-caudal plane. The edge of the sternocleidomastoid is identified and retracted laterally. A blunt Wietlaner, self-retaining retractor is used to maintain the exposure. The retractor is placed superficially on the medial aspect of the incision to prevent injury to the laryngeal nerves, but more deeply on the lateral aspect of the incision.

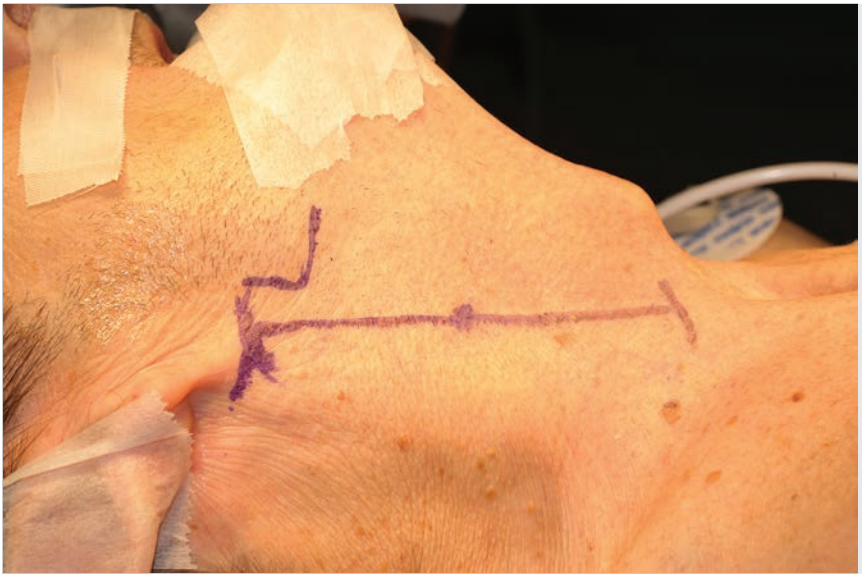

Fig. 35.2 A vertically oriented incision parallels the medial aspect of the sternocleidomastoid muscle. The L-shaped mark denotes the angle of the mandible.

Once deep to the investing fascia, the common facial vein is commonly encountered. This vessel is ligated with two 2–0 silk ties and sharply divided. Dissection continues along the anterior border of the internal jugular vein until the common carotid artery (CCA) is identified proximal to its bifurcation. The carotid sheath is typically opened just above the superior belly of the omohyoid muscle, but in the rare case of a low-lying carotid bifurcation, the omohyoid muscle may be divided with bipolar electrocautery and Metzenbaum scissors. When dividing the omohyoid, a stitch may be placed at the muscle edges to aid in reapproximation at the end of the procedure.

A blunt-toothed, self-retaining retractor can be used to retract the internal jugular vein away from the CCA, although one must be careful to avoid the vagus nerve. As seen in ▶ Fig. 35.3, we now prefer blunt fish-hooks attached to a retaing ring to secure the exposure and prevent nerve damage from retractor use. Once the carotid sheath is opened, a 0 silk tie is placed around the proximal CCA, secured with a Rummel tourniquet, to achieve proximal vascular control.

Carotid Exposure

A strict, "minimum-touch" technique is applied with respect to the CCA to prevent dislodgement of an atheromatous plaque. When the CCA is first identified, 5,000 units of intravenous heparin are administered by anesthesia. Infrequently, dissection along the carotid bifurcation elicits hemodynamic instability. If there is blood pressure lability, then the carotid sinus may be injected with 2 mL 1% Xylocaine (AstraZeneca, Wilmington, DE) via a 25-gauge needle.

The dissection of the carotid complex is completed when the surgeon has isolated the CCA, ECA, and ICA, each of which is then encircled with 0-silk ties or vessel loops. It is critically important to expose the ICA beyond the end of the plaque. The superior thyroid artery is also routinely identified during the anterior cervical dissection and may be controlled with an encircling 2–0 silk tie. The CCA is prepared for proximal control by placement of a Rummel tourniquet that facilitates constriction of the vessel around an intraluminal shunt, if necessary.

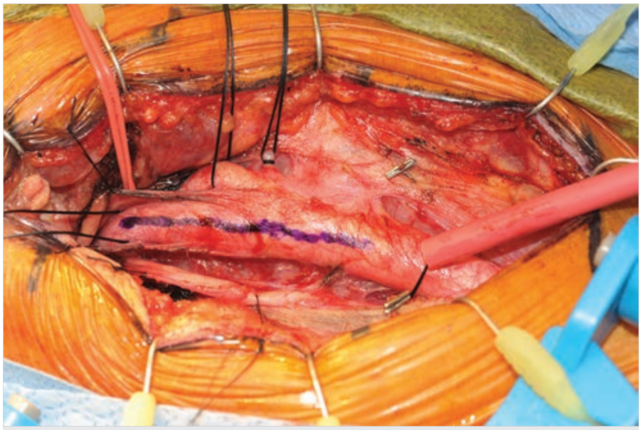

Fig. 35.3 The carotid vessels are isolated and prepared for arteriotomy. Exposure of the internal carotid artery is continued cranially until the surgeon ensures that the intended arteriotomy will extend well above the rostral extent of the plaque. A blue line, demarcating the intended arteriotomy, is useful in preventing a jagged carotid opening, which is difficult to close. Note the presence of a Rummel tourniquet already in its appropriate position, proximally on the common carotid artery.

The carotid plaque is identified by correlating intraoperative visual cues to landmarks noted on and measurements derived from the preoperative angiogram. The yellowish wall of the atherosclerotic carotid artery turns a pinkish-blue distal to the atheroma and it is critical to obtain distal control well beyond the plaque so as not to precipitate arterial thrombi. During exposure of the ICA, a 0-silk suture is passed around it and a Loftus encircling shunt clamp (Scanlan International, St. Paul, MN) is tested around the ICA in case shunting is required. Adequate proximal exposure of the CCA is necessary because the vessel loops are placed 1 cm distal to the area of the DeBakey cross-clamp and the clamp must be placed far enough inferiorly on the CCA so as to facilitate bloodless shunt placement.

35.3.3 Endarterectomy

After adequate proximal and distal vascular control is obtained, a sterile marking pen defines the intended arteriotomy (▶ Fig. 35.3). A bulldog clamp is then used to occlude first the ICA. Next, the DeBakey cross-clamp is used to occlude the CCA. Finally, a second bulldog clamp is applied across the lumen of the ECA. Next, a number 15-blade knife is used to make a stab incision in the proximal CCA. Potts scissors are then used to extend the incision rostrally—taking care to stay in the middle of the lateral exposure of the carotid artery, away from the apex of the bifurcation—until normal vessel lumen is identified.

The arteriotomy and endarterectomy are performed under 3.5x loupe-magnification. Some surgeons use the operating microscope. We use both EEG and SSEP monitoring on these cases. We do not use a shunt in every case, but rather determine whether a shunt is indicated based predominantly on whether there is adequate collateral perfusion of the ipsilateral cerebral hemisphere based on preoperative vascular imaging and whether carotid cross-clamping has any affect on the EEG or SSEP monitoring. Changes in the EEG or SSEP mandate a trial of induced hypertension facilitated by anesthesia; however, if there is no immediate improvement in the EEG or SSEP recording, then an intraluminal shunt is placed.

We use the custom, #10F Loftus CEA shunt of our own design (Integra NeuroCare, Plainfield, NJ). The shunt is first inserted into the CCA and the Rummel tourniquet is tightened around it, then the remaing shunt is passed more proximally into the CCA after the DeBakey clamp has been opened. This method eliminates bleeding from opening the clamp because the tourniquet has already been secured. The distal end is opened to confirm blood flow and to clear any debris from the tubing. The shunt is then inserted in the ICA and secured with the Loftus shunt clamp (▶ Fig. 35.4).[14] A handheld Doppler is applied to the tubing to auscultate flow.

Plaque removal begins with a Freer elevator (Sklar Instruments, West Chester, PA) or Scanlan plaque dissector (Scanlan Instruments, St. Paul, MN), either of which may be used gently to develop a cleavage plane between the atheromatous plaque and intimal layer of the vessel wall (▶ Fig. 35.5). Dissection begins at the rostral aspect of the plaque and continues caudally in a circumferential manner. Meticulous dissection prevents breaching through the lateral aspect of the vessel wall. If the plaque extends proximally into the CCA and no feathered edges

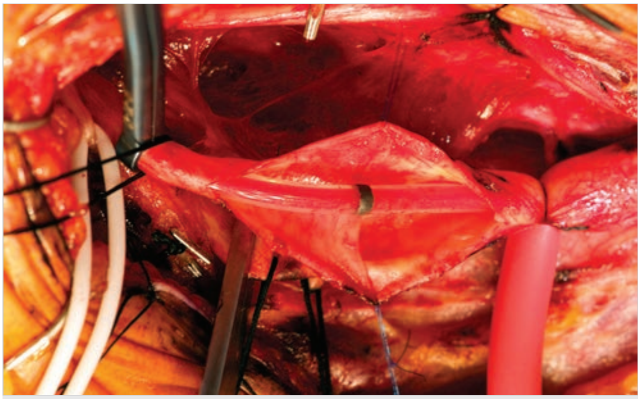

Fig. 35.4 The Loftus shunt is secured into place within the lumen of the common carotid artery by the Rummel tourniquet. Care must be taken to place the black marking band at the center of the arteriotomy to ensure that the shunt does not migrate during plaque dissection and patch grafting. Prior to its insertion into the internal carotid artery, the shunt is opened and bled for evacuation of debris.

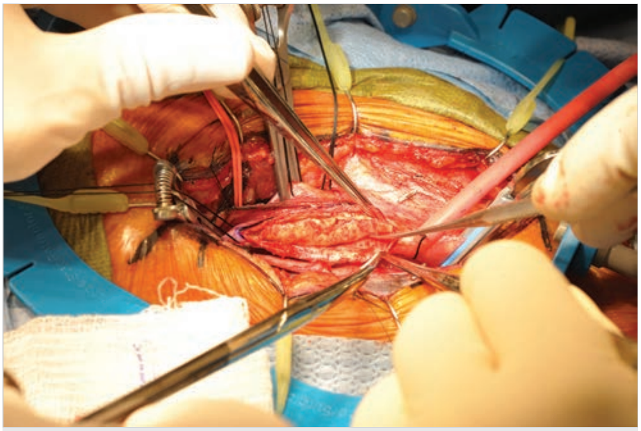

Fig. 35.5 The plaque is dissected off the intimal wall of the internal carotid artery with either a Freer elevator or number four Penfield microdissector.

are readily identified, then the plaque is transected sharply with either a number 15-blade knife or tenotomy scissors. If the dissection is taken superiorly enough, then the atheromatous plaque usually feathers distally into the ICA and can be easily removed. In some cases, however, at the distal endpoint the plaque may leave a “shelf” with tattered edges that must be cleaned, the edges of which must be tacked down with 6–0 Prolene (Ethicon Inc., New Brunswick, NJ) sutures to prevent arterial dissection. After separation of the plaque from the ICA and the CCA, the remaining plaque is secured with vascular forceps and removed from the ECA. If there is any question of residual plaque, then a separate arteriotomy and primary repair of the ECA should be undertaken.

After plaque removal, the arterectomy site is irrigated with heparinized saline to detect any residual atheromatous fragments that can then be removed with microscopic, ring-tipped forceps from the Scanlan Loftus set. If tacking sutures are required in the distal ICA, double-armed 6–0 Prolene sutures are inverted vertically, from the inside of the vessel outward, so the sutures traverse the intimal edge and are tied outside of the adventitial layer.

35.3.4 Carotid Closure

It is our practice to use a synthetic patch graft in all cases. We prefer the Hemashield collagen-impregnated Dacron patch graft (Hemashield: Maquet Getinge Group, Rastatt, Germany). Advantages of this patch are: (1) it is quickly and easily shaped to size with scissors; (2) it does not require preclotting or special handling; (3) there is little to no leakage from the required tacking suture holes; and (4) standard, 6–0 Prolene suture may be used. Other surgeons describe primary arteriotomy closure.

The patch material is placed over the surgical field and cut according to the length of the arteriotomy. The ends are trimmed and tapered to a point with Metzenbaum scissors. Each end of the patch is anchored to the arteriotomy with double-armed 6–0 Prolene sutures (▶ Fig. 35.6). The suture line along the medial wall of the vessel is closed first with a running, nonlocking stitch. The lateral wall is closed next using the same running, nonlocking technique, but anchored both at the rostral and caudal ends of the arteriotomy such that the sutures lines meet at the midpoint of the arteriotomy. The sutures should be evenly spaced and placed within millimeters of the arterial edge along the length of the arteriotomy to create a water-tight vascular closure. Several millimeters of the vessel are left unsewn along the lateral wall of the vessel, ensuring adequate space is left through which the shunt may be removed. The shunt is extracted by securing it with two parallel straight mosquito clamps and then cutting the shunt tubing in half, allowing it to be removed easily in two pieces.

The final sutures are then placed once the shunt has been removed, but are not secured until the arterectomy site is flushed out by temporarily opening and closing the clamps in sequence of ICA, ECA, and CCA. Heparinized saline is introduced into the arterial lumen to evacuate air prior to securing the final surgeon’s knot across the arteriotomy. When the vessel is closed, the vascular clamps are removed in the reverse sequence in which they were initially placed—ECA, CCA, and finally, 10 seconds later, from the ICA. This sequence of clamp removal ensures that any atheromatous debris or air emboli are flushed into the extracranial circulation rather than into the intracerebral circulation. Once all the clamps are removed, the suture lines are inspected for any leakage, the majority of which may be addressed simply with the application of surgical gauze and light pressure. If needed, single throw 6–0 Prolene sutures are used to buttress the arterial closure at arterial leak points. The repair is then lined with Surgicel (Ethicon Inc., New Brunswick, NJ) and the handheld Doppler is used to confirm vessel patency. The fish hook retractors are then carefully removed and the wound is closed in anatomic layers. A Hemovac drain (Davol Inc., Murray Hill, NJ) is placed within the carotid sheath prior to its primary closure. The platysma is then gently approximated to optimize cosmesis. Running subcuticular stitches are then placed to approximate the skin edges and Steri-StripsTM (3 M Company, St. Paul, MN) are placed along the incision line. The Hemovac is typically removed on postoperative day 1.

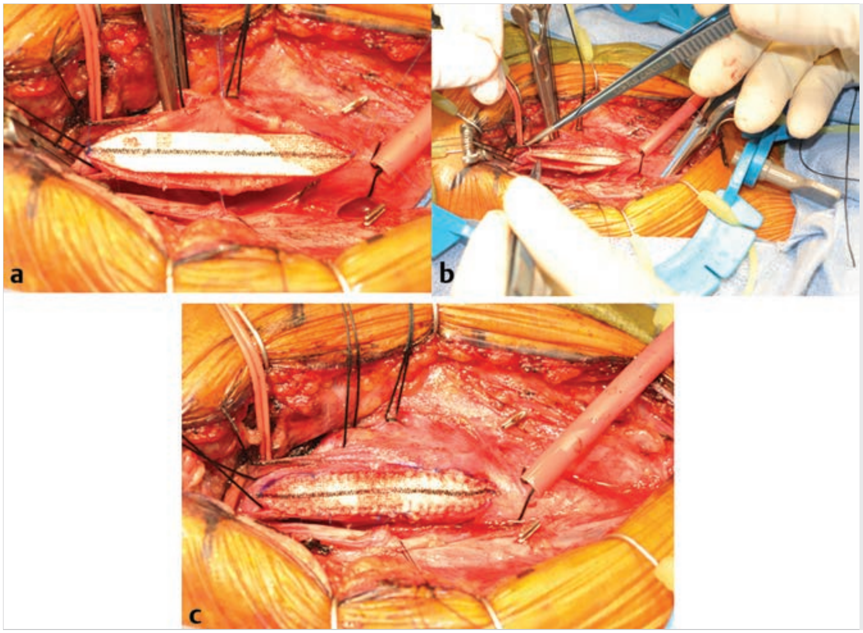

Fig. 35.6 **(a)** After cleansing the plaque bed, the arteriotomy is closed with a Dacron patch, beginning at the distal apex where it is secured with a 6–0, double-armed monofilament suture. Each arm is placed through the patch and then through the vessel wall from the lumen to the outside. **(b)** The medial wall of the arteriotomy has been closed first with a running, nonlocking stitch and now the lateral vessel wall suture line is starting. **(c)** Completed arterial repair: the lateral wall has now been closed in the same running, nonlocking fashion—initiated at both the rostral and caudal limits of the arteriotomy and converging at the midpoint of the arteriotomy.

35.4 Postoperative Care

Patients are monitored in the recovery room or are transferred directly to the neurological intensive care unit. Blood pressure and neurological function are the most important parameters to monitor. During the first 12 to 24 hours postoperatively, blood pressures are frequently labile and the goal is to maintain the systolic blood pressure between 100 and 160 mm Hg. It is critical to identify and treat hypertension promptly as many CEA patients demonstrate cerebral autonomic dysregulation that predisposes them to reperfusion hemorrhage. In addition, cardiac monitoring with telemetry is recommended, as myocardial infarction following CEA may occur.

Patients are transferred to the ward after 24 hours of neurologic and hemodynamic monitoring and are typically discharged home the following day. Currently, there are no evidence-based guidelines for uniform postoperative surveillance. Our routine surgical follow-up consists of a wound check at 4 weeks and a carotid duplex ultrasound at 3 months to confirm carotid patency.

Stroke is the second most common cause of death following CEA and is attributable to multiple factors, including plaque emboli, platelet aggregates, improper flushing, poor cerebral protection, and relative hypotension. Any postoperative neurological deficit, including TIA, should be addressed aggressively with immediate CTA. If an acute occlusion of the carotid artery is identified, then the patient should be taken back to the operating room for exploration and to reestablish patency.

Cervical hematoma may cause acute airway compromise and expanding neck hematomas in the postoperative period should be closely monitored. Injuries to the hypoglossal, facial (marginal mandibular branch), recurrent laryngeal and accessory nerves are also known complications of anterior cervical dissections.

While radiographic and ultrasonic evidence of residual carotid stenosis is not uncommon, the rate of clinically significant, recurrent carotid stenosis after CEA that requires intervention is extremely low and continues to decline with refinement in surgical technique. It has been estimated that, with the placement of a patch graft at the time of the index CEA, the incidence of recurrent carotid stenosis is 5% at 2 years.[15] In our series, since adopting the universal patch repair, neither acute perioperative nor delayed carotid restenosis has been observed.[16]

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36 Superficial Temporal Artery to Middle Cerebral Artery Bypass

Ziad A. Hage, Sepideh Amin-Hanjani, and Fady T. Charbel

Abstract

Superficial temporal artery–middle cerebral artery (STA–MCA) bypass grafting is the mainstay of cerebral revascularization surgery and is used for flow augmentation and flow replacement. Neurovascular surgeons should aspire to have this and other bypass operations as part of their technical expertise. A catheter angiogram is an essential component of the preoperative evaluation, including selective external carotid injections to evaluate the caliber and course of the STA branches on the affected side. The operation involves harvesting the STA and protecting it in a papaverine-soaked cottonoid. A craniotomy is performed and a recipient MCA branch is selected based on size, location, and orientation and dissected free from the surrounding arachnoid. All the perivascular tissue is removed from the distal end of the STA and the STA–MCA anastomosis is performed in an end-to-side fashion. Blood flow measurements performed on the STA before, and the bypass after, completion are useful to confirm the patency and function of the bypass.

Keywords: STA–MCA bypass, flow augmentation, flow replacement, cut flow, anastomosis

36.1 Introduction

Cerebral revascularization can be performed through a variety of extracranial–intracranial bypass operations that use several different donor and recipient arteries, interposition grafts, and anastomotic techniques.[1,2,3,4] The choice of bypass option is dependent on multiple factors, including the goals of the operation and the availability and accessibility of donor and recipient vessels.[1,2,3,4,5,6,7] The superficial temporal artery–middle cerebral artery (STA–MCA) bypass, however, is a mainstay of cerebral revascularization surgery and is described here.[1,4]

36.2 Patient Selection

The two main indications for extracranial to intracranial bypass are flow replacement for treatment of complex aneurysms or tumors, which requires vessel sacrifice, and flow augmentation for treatment of cerebral ischemia in those demonstrating misery perfusion.

Sacrifice of the parent vessel may be required for definitive management of giant or complex aneurysms or to allow complete excision of skull-base tumors.[8,9,10,11] Although carotid sacrifice may be tolerated, stroke can occur in up to 30% of patients. Endovascular balloon test occlusion combined with hypotensive challenge and neurological, electrophysiological, and blood flow testing can be used to select patients who will not tolerate carotid sacrifice and who may benefit from surgical revascularization.[2,3] Fusiform or unclippable aneurysms of distal vessels, such as the MCA or its branches, would typically require revascularization because collaterals to such terminal vessels are inadequate.[2,3]

Revascularization for patients with ischemic disease is controversial and case selection is individualized. Our indications for STA–MCA bypass are failure of maximal medical therapy (adequate antiplatelet or anticoagulant regimen, combined with modification of vascular risk factors), symptoms (stroke or transient ischemic attacks) concordant with radiographic findings, compromised cerebrovascular reserve as demonstrated by multimodal magnetic resonance imaging (MRI) using quantitative MR angiography performed with the noninvasive optimal vessel analysis software (VasSol, Inc., Chicago, IL)[12] before and after acetazolamide challenge, and functional MRI with regional BOLD sequences using a multivessel territory task paradigm, as well as global BOLD before and after a CO_2 challenge.[2,3,13] Direct STA–MCA bypass is also an option in the treatment of symptomatic adult moyamoya disease.[7]

36.3 Preoperative Preparation

In addition to the disease-specific workup that pertains to the indication for the STA–MCA bypass, a catheter angiogram is obtained to delineate the intracranial lesion.[4] Selective external carotid injections evaluate the caliber and course of the STA branches on the affected side. If there is concern regarding the adequacy of the STA, alternative bypass strategies using interposition grafts (saphenous vein or radial artery) anastomosed to the STA trunk or the cervical carotid can be entertained. Duplex mapping of the saphenous veins is useful to reveal the size of the veins and their course. If radial artery harvesting is contemplated, an Allen's test should be performed while monitoring the oxygen saturation on the thumb.

Patients take 325 mg acetylsalicylic acid the night prior to the surgery.[4] If they are on warfarin, it is stopped and they are started on heparin, which is withheld 6 hours prior to surgery because antiplatelets are administered. Arterial line and central venous access are routinely obtained. Perioperative antibiotics are administered.

For ischemia cases, normovolemia, normocapnia, and normotension are maintained throughout the surgery.[4] For aneurysms, cerebrospinal fluid drainage via a lumbar drain can be used for brain relaxation to avoid the need for intravenous diuretics (furosemide), hyperosmolar agents (mannitol), or hyperventilation. Scalp electrodes for electroencephalographic monitoring are placed outside the surgical field. This allows for induction of burst suppression during temporary vessel occlusion. During temporary vessel occlusion mean arterial blood pressure is also raised, with the goal of achieving values 25% above baseline.

36.4 Operative Procedure (Video 36.1 and 36.2)

36.4.1 Positioning

The head is fixed in the lateral position with a four-pin Sugita head holder (▶ Fig. 36.1). A shoulder roll may be needed in patients with restriction in cervical lateral rotation. If intraoperative angiography is desired, a radiolucent frame (Mizuko America, Inc., Beverly, MA) is used and the right groin is prepared and draped for placement of a femoral angiography catheter. In the case of saphenous vein or radial artery harvesting, the appropriate lower (based upon size and length) or upper limb (the nondominant forearm) is also prepared and draped. For the lower limb, generally the thigh vein is used. For radial artery, the area of the ventral forearm extending from the hand to the antecubital fossa is prepared.

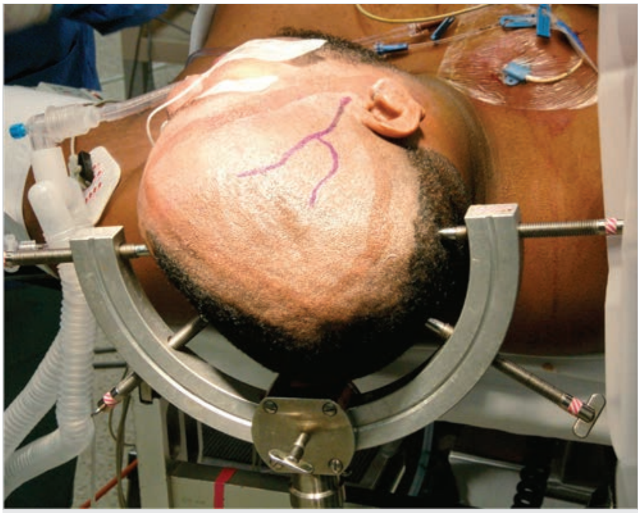

Fig. 36.1 Positioning for a right superficial temporal artery (STA)–middle cerebral artery bypass. The posterior and anterior branches of the STA are traced with Doppler and marked on the scalp as shown.

The scalp is shaved and a Doppler is used to map the STA starting at the level of the zygoma, as well as both anterior and posterior branches of the vessel (▶ Fig. 36.1).

36.4.2 Skin Incision

The skin incision generally overlies the STA trunk just anterior to the tragus in the region of the zygoma and extends along the course of the posterior branch in a linear fashion.[4] In the case of a bypass for ischemia, a linear incision of this nature is adequate. If the posterior branch is inadequate, an incision could be placed directly over the anterior branch instead, but frequently this course will carry the incision onto the forehead. In such cases it might be preferable to create a semicircular incision behind the hairline (▶ Fig. 36.2) and expose the anterior branch from the undersurface of the skin flap. This is also used when a posterior branch is initially exposed but appears to be of poor quality or caliber—the linear incision is merely curved forward and converted into a skin flap, which allows dissection of the anterior branch. In the case of bypass for aneurysm, the linear incision over the posterior branch of the STA is curved forward in a semicircular fashion resembling the standard pterional incision, to allow access for the required craniotomy.

36.4.3 Superficial Temporal Artery Dissection

The initial incision through the epidermis and dermis is made with a Colorado microneedle tip monopolar cautery (Stryker Leibinger, Kalamazoo, MI), set on 8, along the midpoint of the projected course of the STA branch. We perform this under loupe or microscopic magnification with the surgeon and assistant seated. Opening the skin in this fashion limits bleeding from the skin edges, whereas the low cautery setting helps to prevent skin edge necrosis or poor wound healing. Once subcutaneous tissue is encountered, a blunt tip, fine curved snap is used to dissect down to the STA. Once the vessel is visualized the snap is used to dissect proximally in the loose areolar plane above the vessel. The Colorado tip is then used to open the skin

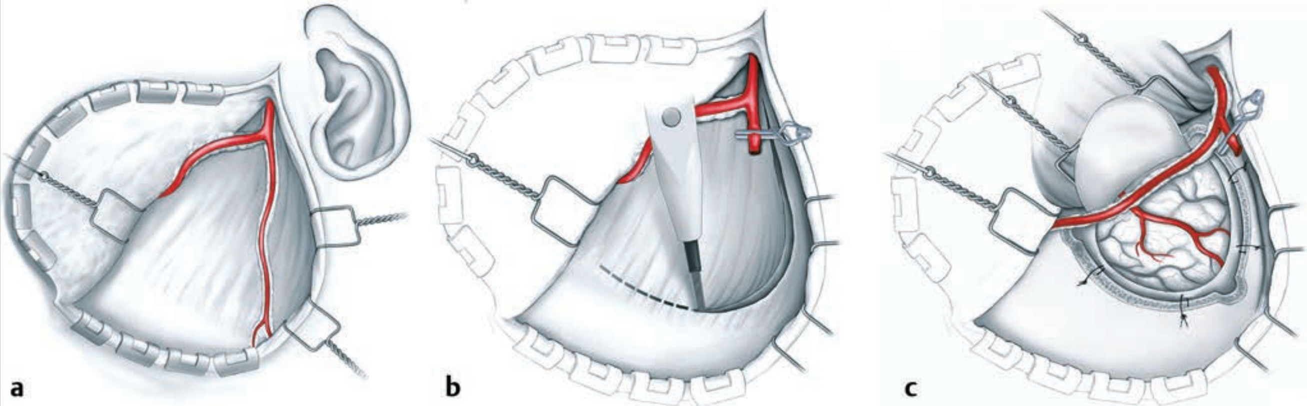

Fig. 36.2 Steps in performing the initial exposure for a superficial temporal artery–middle cerebral artery (STA–MCA) bypass. **(a)** The STA trunk and branch are exposed and dissected free with their surrounding cuff of tissue. **(b)** The temporalis muscle is divided and retracted anteriorly or in a T-shaped/cruciate fashion, maintaining the STA in continuity. **(c)** Burr holes are placed and the craniotomy flap is elevated, a pterional flap for aneurysm exposure or more limited craniotomy for access to cortical vessels in ischemia cases as shown. The dura is opened and flapped anteriorly or in a cruciate fashion.

to the tip of the snap as sequential dissection is performed, until the main trunk of the STA is reached. This procedure is continued distally.

The goal is to dissect about 8 to 10 cm of STA. Once exposed, Bovie electrocautery with a coated shaft at a setting of 25 to 30 is used to divide the tissue around the STA and allow it to be lifted from the underlying temporalis muscle fascia. The dissected STA and its surrounding tissue are then wrapped in a papaverine-soaked cottonoid to alleviate spasm induced by the mechanical manipulation of the vessel.[4]

36.4.4 Craniotomy

Self-retaining fishhook retractors are placed at the skin edges with the STA within its protective cottonoid reflected to one side or the other. Bovie electrocautery is used to incise the temporalis fascia and muscle. For aneurysms, the muscle is cut along the line of the scalp incision and reflected anteriorly with the skin flap (▶ Fig. 36.2**).** For ischemia, the muscle can be opened symmetrically under the linear incision in a T-shape or cruciate fashion, with each quadrant of muscle retracted anteriorly or posteriorly with hooks.

For aneurysms, a standard frontotemporal pterional bone flap is elevated with removal of the sphenoid ridge extradurally. For ischemia, a burr hole is placed with a Midas (Medtronic, Inc., Fort Worth, TX) round drill bit below the proximal and distal aspect of the course of the vessel and a circular craniotomy created with the Midas (Medtronic) B1 drill bit (▶ Fig. 36.2). Thin strips of Gelfoam (Pfizer, Inc., New York, NY) or Surgicel (Johnson and Johnson, Somerville, NJ) are packed under the bone edges and the dura is tacked extensively around the margins of the craniotomy to stop and prevent epidural bleeding.

The dura is opened as a semicircular flap, which is retracted anteriorly (▶ Fig. 36.2**,** ▶ Fig. 36.3) or alternatively in a cruciate fashion with additional cuts placed to form multiple triangular flaps, which are tacked backward, exposing the cortex.[4] In moyamoya disease, dural arteries should be preserved because these are potential sources of collateralization.

36.4.5 Preparing the Recipient Artery

The microscope is used for all intradural aspects of the procedure. For ischemia cases, the cortical surface is examined for a suitable recipient cortical MCA branch. The most important consideration is the size of the recipient—a vessel of 1.5 mm or greater is optimal. Other considerations are the location (away from the craniotomy edges) and orientation of the vessel (a tangential orientation of the vessel from the upper left to the lower right is optimal for a right-handed surgeon because it creates the most natural angle for placement of sutures during the anastomosis). The arachnoid over the potential recipient vessel is opened with an arachnoid knife, fine forceps, and microscissors and a 1 cm length is prepared for anastomosis. Small perforators emanating from the artery must be coagulated with bipolar cautery so the artery can be elevated from the brain surface for placement of a rubber dam underneath the vessel. Larger perforators can be spared by occlusion with temporary vessel clips during the anastomosis. A piece of Gelfoam (Pfizer, Inc., New York, NY) is placed under the rubber dam to elevate the artery out of its sulcus and a papaverine-soaked cotton ball is applied to alleviate spasm.

For aneurysm cases, the sylvian fissure is opened widely to expose the MCA bifurcation and its branches. A segment of the second or third portions of the middle cerebral artery can then be prepared as the recipient site.

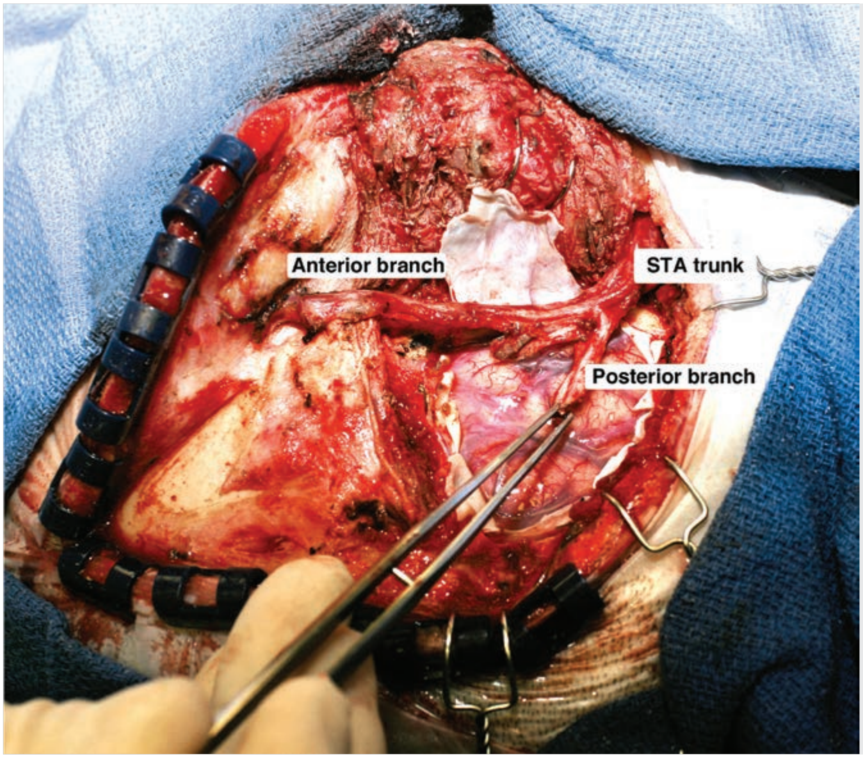

Fig. 36.3 The completed exposure for a right-sided bypass for ischemia.

36.4.6 Preparing the Donor Artery

The brain is covered with cottonoids and the STA branch is uncovered. A sharp curved snap is used to dissect the artery from the surrounding cuff of tissue proximally to create a site for placement of a temporary clip. The ideal site for temporary clipping is distal to the take-off of the unused branch of the STA; this allows continued flow through the STA into the unoccluded branch, reducing stagnant flow and the risk of thrombosis proximal to the temporary clip. If it is necessary to sacrifice the unused branch to adequately mobilize the STA, the branch should be divided with a temporary clip on the proximal stump. This will allow the stump to be used for back-bleeding and venting of air following anastomosis.[4]

The STA is next dissected at its most distal aspect. Sugita temporary clips (Mizuho America, Inc., Beverly, MA) are placed proximally and distally and the vessel and its cuff are transected distally (▶ Fig. 36.4). The distal end of the STA is coagulated and the temporary clip removed. A blunt-tip needle is used to flush the STA with heparinized saline (10 U heparin/mL) through its cut end. The proximal temporary clip is opened so the flush goes proximal to the clip. Once flushed, the required length of STA to reach the anastomotic site without tension is gauged and this length is marked on the vessel with a marking pen. The vessel is dissected free of its cuff of tissue to a point 2 cm proximal to this.[4] The "cut flow" in the donor artery can then be measured (▶ Fig. 36.4). The STA is flushed with heparinized saline following this maneuver.

36.4.7 Performing the Anastomosis

The STA–MCA anastomosis is performed in an end-to-side fashion. The STA is cut at a 45 degree angle and a fish mouth created to enlarge the opening (▶ Fig. 36.5). A marking pen is used to color the outer wall of the vessel which allows the lumen to be seen more easily and the line of incision on the recipient vessel is also marked for the same purpose.

Temporary mini Sugita clips (Mizuho America, Inc., Beverly, MA) are placed proximally and distally on the recipient vessel, which is then incised with an ophthalmic blade. The fish-mouthed donor STA is laid next to the recipient and the vessel is opened to the exact length necessary with microscissors (▶ Fig. 36.5, ▶ Fig. 36.6). The opened vessel lumen is flushed with heparinized saline.[4]

Nylon suture (10–0) is used to place anchoring sutures at the apices of the incision (▶ Fig. 36.5, ▶ Fig. 36.6). We place the stitch at the "toe" of the anastomosis first. Sutures should be passed through the recipient vessel from inside to outside

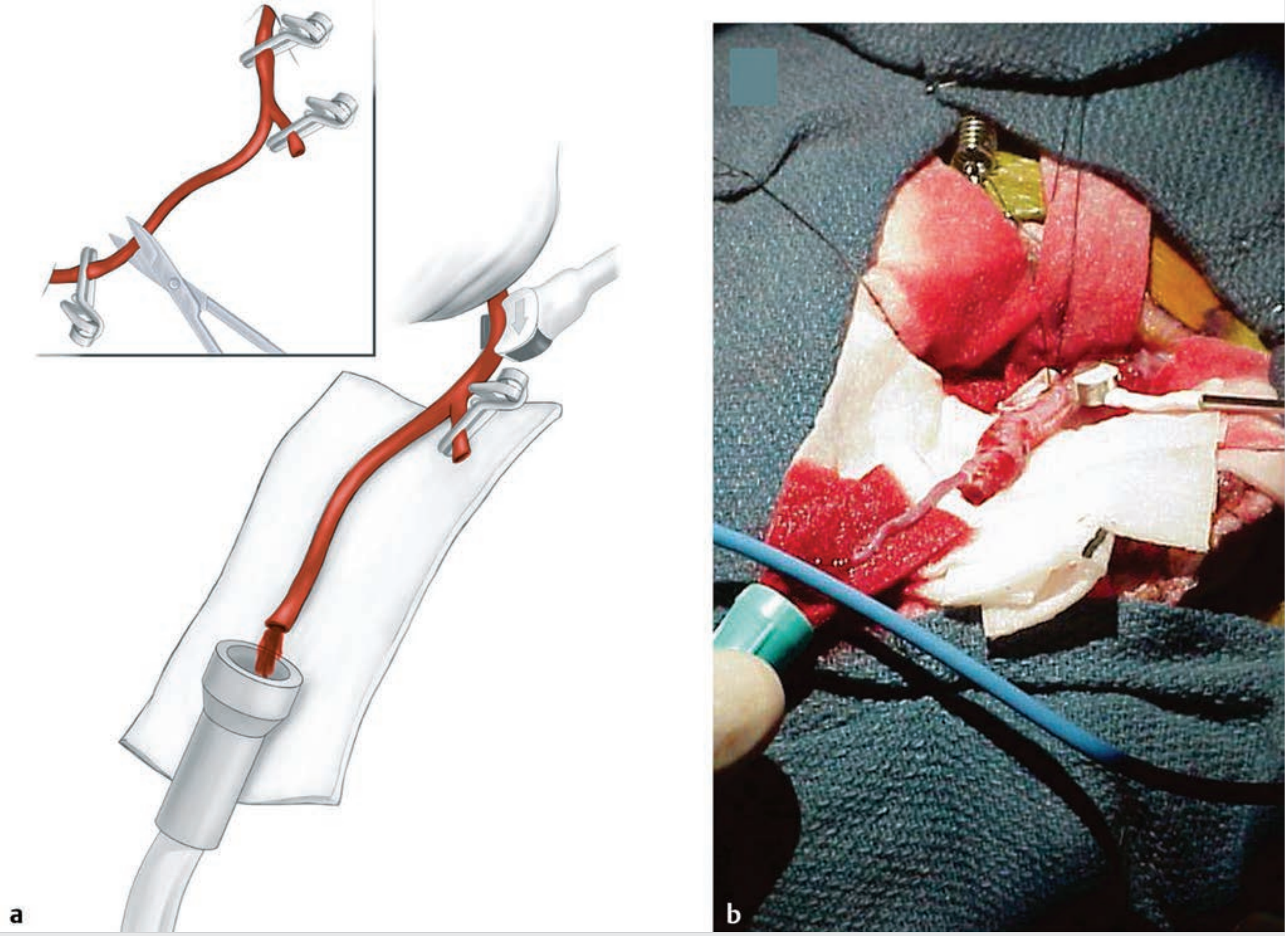

Fig. 36.4 Measurement of "cut flow" through the open end of the superficial temporal artery (STA). **(a)** Cutting the STA under temporary occlusion and then measuring the flow with a Doppler with the temporary clip removed. **(b)** Intraoperative photograph showing the method of measurement.

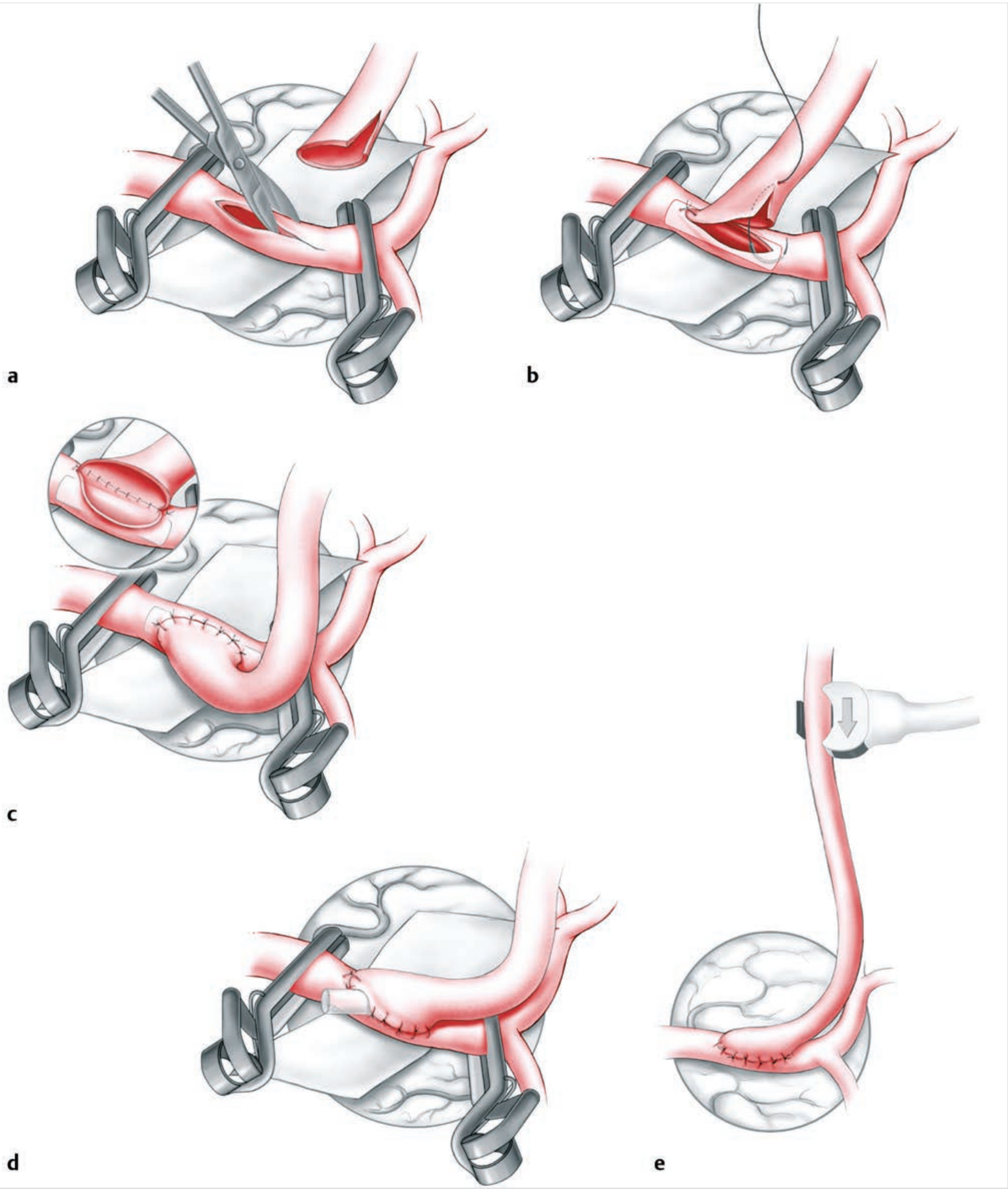

Fig. 36.5 Steps in performing the anastomosis. **(a)** The superficial temporal artery (STA) is cut and a fish mouth of desired length is created. Temporary clips are placed on the recipient vessel and the arteriotomy is created with a sharp blade and extended with microscissors. **(b)** A Silastic stent (Dow Corning Corp., Midland, MI) is placed within the recipient vessel and 10–0 nylon sutures are placed at the apices. **(c)** Interrupted sutures as shown or running suture, are placed along one side of the anastomosis and the suture line is examined from the internal aspect of the lumen (inset) prior to proceeding with the other side. **(d)** The stent is removed prior to placement of the final stitches. **(e)** Flow is measured in the completed bypass graft with a flow probe.

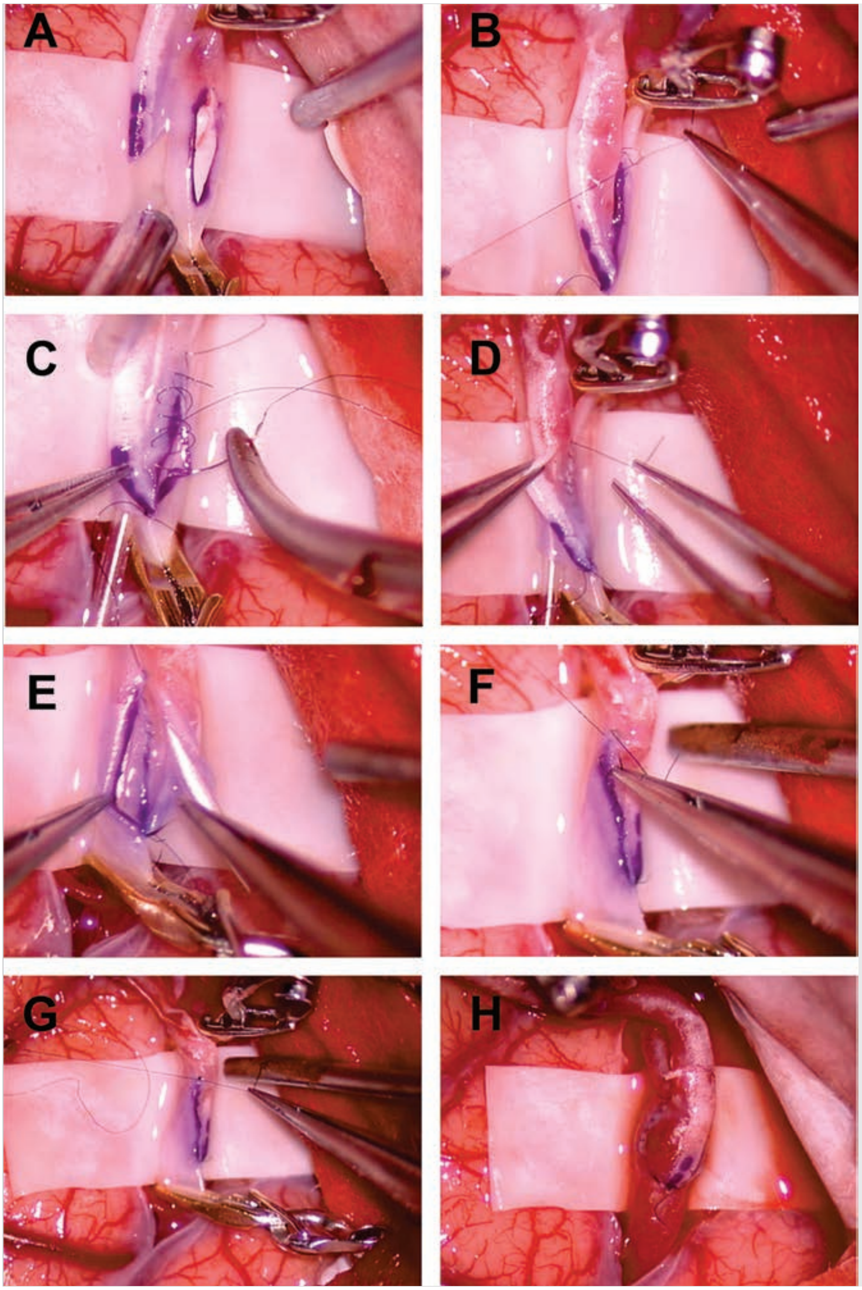

Fig. 36.6 Steps in performing an anastomosis using running suture. **(a)** The superficial temporal artery (STA), colored with a marking pen, is laid next to the recipient vessel, also marked, to create an arteriotomy of the desired length. **(b)** The apical "toe" stitch has been placed and the apical "heel" stitch is next placed and tied. **(d)** A running suture is placed on one side of the anastomosis leaving the individual loops loose to be tightened sequentially before tying the suture. **(d)** Even tension along the entire suture line is achieved. **(e)** The inside of the arteriotomy is examined to inspect the suture line to ensure that there are no technical errors. **(f)** Sutures are placed on the other side of the anastomosis and the loops are tightened. **(g)** The suture is tied to the end of the apical stitch after the loops have been tightened. **(h)** After completing the anastomosis, temporary clips are removed to open the bypass.

to reduce injury to the vessel endothelium. Once the donor STA has been anchored, interrupted sutures are placed on each side of the anastomosis at close intervals (▶ Fig. 36.5, ▶ Fig. 36.7). Three knots are enough and all sutures are tied except the penultimate suture, which is left free to allow room for good visualization as the final suture is placed. Continuous suture technique can also be used. This involves leaving short loops of suture along the entire length of the vessel (▶ Fig. 36.6), which are tightened sequentially just prior to tying the suture.

During suturing, the thin walls of the recipient artery tend to collapse together and inadvertent suturing of the opposite wall is a distinct concern. To prevent this, a Silastic stent (Dow Corning Corp., Midland, MI) is placed within the recipient vessel and removed just prior to the final sutures (▶ Fig. 36.5 and ▶ Fig. 36.7). The lumen is flushed with heparinized saline prior to tying the final suture. Once the anastomosis is complete, the temporary clips on the recipient artery are released and then the proximal clip on the STA is removed. Blood flow in the STA is measured, which confirms patency and function of the bypass (▶ Fig. 36.5). We have found that a "cut flow index" (ratio of the bypass flow to the initial cut flow) is a sensitive predictor of bypass function. Intraoperative angiography is rarely necessary if direct flow measurements are performed.[2,11]

36.4.8 Closure

The dura is loosely sutured or not closed and the dural opening is covered with a piece of Gelfoam (Pfizer, Inc., New York, NY). The bone is replaced, but the burr hole is enlarged to accommodate

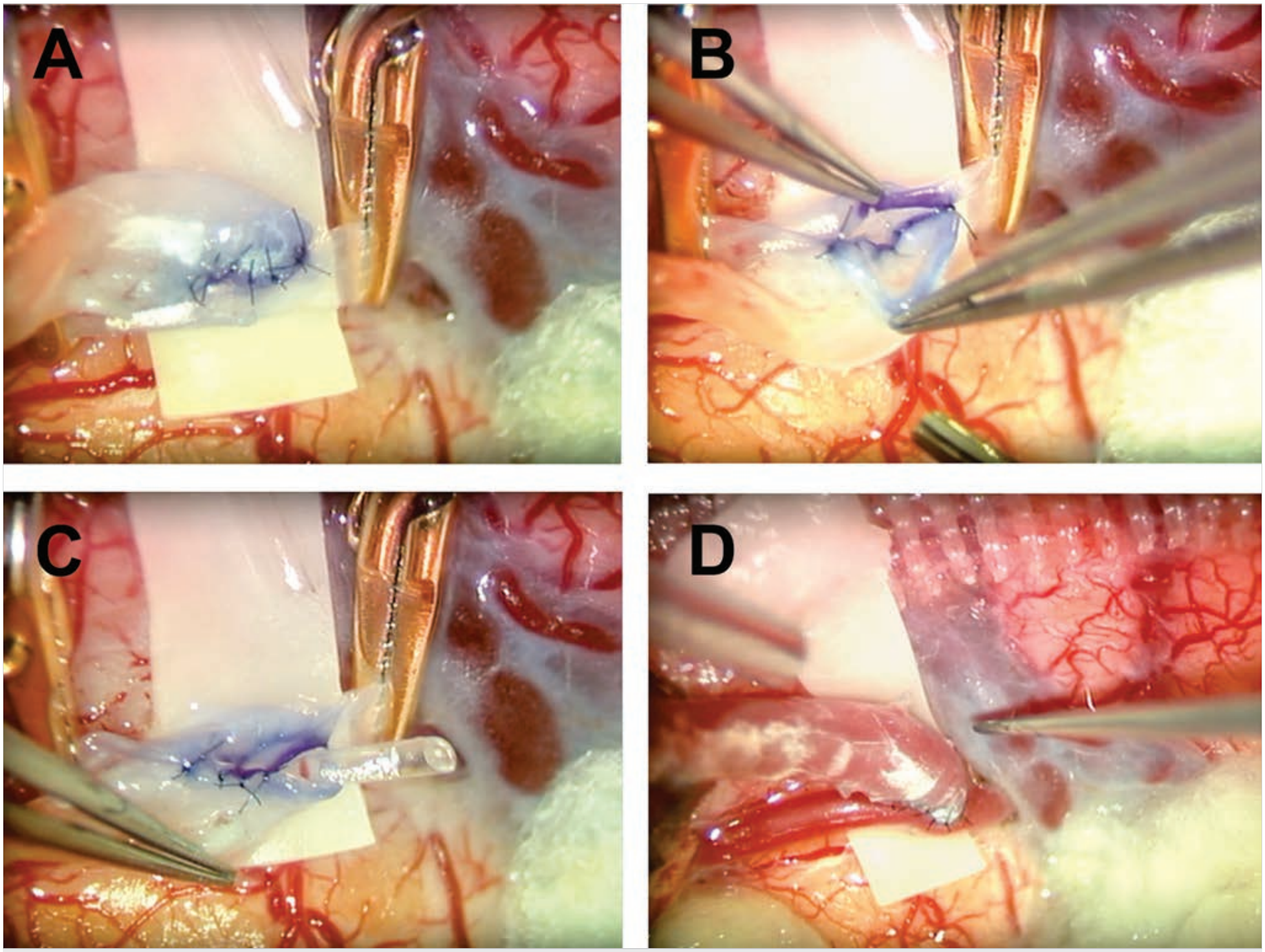

Fig. 36.7 Steps in performing an anastomosis using interrupted sutures. **(a)** Interrupted sutures placed on one side of the anastomosis at uniform intervals. **(b)** The inside of the arteriotomy is examined to inspect the suture line before proceeding with the other side of the anastomosis. **(c)** The intraluminal stent is removed before the final sutures are placed. **(d)** The temporary clips are removed to open the bypass.

the STA, avoiding any kinking or pressure on the vessel. The muscle is reapproximated loosely and the skin is closed with care to avoid injury to the proximal STA.

36.5 Postoperative Management Including Possible Complications

Patients are resumed on acetylsalicylic acid, 325 mg daily, starting immediately postoperatively. Patients are observed in the intensive care unit postoperatively and are kept well hydrated. Hypotension is avoided. Bedside monitoring of bypass function in the immediate perioperative period can be performed with Doppler of the STA trunk. Pressure over the temple region (by nasal oxygen cannula or glasses) is avoided. Baseline angiography is performed postoperatively.[4]

Potential postoperative complications include epidural hematoma or wound infection. Postoperative graft occlusion is rare, given that bypass function can be well assessed intraoperatively with flow measurement and graft revision performed at that time if necessary.

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37 Indirect Bypasses for Moyamoya Disease

Edward Smith

Abstract

Moyamoya is a progressive intracranial arteriopathy with a high risk of stroke if left untreated. Surgical revascularization markedly reduces this risk, particularly in children. Operative approaches should be considered whenever there is a clear radiographic evidence of advanced moyamoya, even if asymptomatic. Selection of direct or indirect bypass remains controversial, although data and American Stroke Association Guidelines support the use of indirect operations in younger children. Meticulous surgical technique, with careful adherence to perioperative protocols can mitigate operative risk. Overall, surgical treatment of moyamoya can confer long-lasting, significant protection from stroke. This chapter reviews the surgical technique and perioperative care of children undergoing indirect revascularization for moyamoya.

Keywords: moyamoya, revascularization, pial synangiosis, indirect, stroke

37.1 Introduction

Moyamoya is an arteriopathy of the intracranial internal carotid arteries (ICAs), characterized by progressive narrowing of the intradural ICA and proximal middle and anterior cerebral arteries with concomitant development of a network of collateral vasculature likened to a "puff of smoke" when seen on a catheter angiogram. Moyamoya syndrome refers to patients with an identified cause, such as cranial irradiation. The syndrome may be unilateral. Moyamoya disease is bilateral and idiopathic. The etiology of the disorder remains under investigation, with data suggesting that different genetic and epigenetic influences contribute to initiating a process that ends with a shared common pathology of smooth muscle cell overgrowth in the media of the vessels, leading to stenosis.[1] This stenosis ultimately causes critical reductions in blood flow to the brain, leading to cerebral ischemia and hemorrhage.

Once moyamoya is diagnosed, evidence supports surgical revascularization as the primary therapy, even in most asymptomatic children.[2,3,4] Without intervention, there is an annual 13% risk of ischemic stroke coupled with a 7% risk of hemorrhage.[5] Indirect revascularization is the more common intervention in children, a surgical procedure (such as pial synangiosis), in which a pedicle of vascularized tissue is grafted to the brain and used as a source of new blood supply to reduce the risk of stroke. Done at high volume centers, this procedure can reduce the 5 year risk of stroke to 4.7%; a near 20-fold reduction.[2,6]

37.2 Diagnosis

Once suspected, the evaluation of moyamoya should start with magnetic resonance imaging (MRI) and magnetic resonance angiography. Many patients will show watershed infarcts, evidence of narrowed branches of the ICA and up to 81% of symptomatic patients will have the ivy sign—sulcal hyperintensity on FLAIR imaging indicative of slow blood flow.[7] Angiograms should include injections of both carotid (both internal and external) and both vertebral arteries. Important features to note include the presence of spontaneous transdural collateral vessels, particularly those arising from vessels contained within the surgical field such as the superficial temporal artery (STA) and the middle meningeal artery.

Typically, patients are started on aspirin (81 mg daily) and counseled to avoid dehydration and hyperventilation. Referral to a specialist in moyamoya, ideally in a multidisciplinary practice at a high-volume center is an appropriate next step.[2,8]

37.3 Indications and Contraindications

The primary objective of surgery is to create a new vascular supply to the brain to correct the ischemia caused by the arteriopathy. Guidelines from the Japan Ministry of Health and Welfare regarding indications for surgical treatment of moyamoya state the following: "In the cases with (1) repeated clinical symptoms due to apparent cerebral ischemia or (2) a decreased regional cerebral blood flow, vascular response and perfusion reserve, based on the findings of a cerebral circulation and metabolism study, surgery is indicated."[9] In the United States, the American Stroke Association guidelines are similarly broad, suggesting that indications for revascularization surgery include "progressive ischemic symptoms or evidence of inadequate blood flow or cerebral perfusion reserve in an individual without a contraindication to surgery."[10] Consequently, indications for surgery in many centers include: (1) radiographic evidence of moyamoya and either (2) symptomatic moyamoya at any Suzuki stage, or (3) asymptomatic moyamoya with Suzuki II–VI and/or evidence of progressive radiographic changes suggestive of ischemia (such as FLAIR changes or worsening perfusion on arterial spin labeling MRI).[2,3,8]

Contraindications include: (1) those patients with unclear diagnoses, (2) asymptomatic hemispheres with low Suzuki stages (I–II) without clear evidence of ischemia, or (3) patients medically unfit for the operating room (such as those with severe cardiac or pulmonary disease).

37.4 Selection of Approach

Surgical revascularization is typically accomplished using branches of the external carotid artery (which are unaffected by moyamoya) as donor vessels. Indirect approaches rely on the growth of new vessels from a transplanted supply, usually the STA, although any vascularized tissue (such as muscle, pericranium, omentum, or dura) has potential to work.[11] There are a bevy of named indirect approaches, including encephaloarteriodurosynangiosis, pial synangiosis, encephalomyosynangiosis, dural inversion, and multiple burr holes.[12] Indirect approaches have the advantages of working in any age, not being limited by donor vessel size and providing long-term durable revascularization (**Video 37.1**). The main limitation is that indirect approaches require weeks to grow new vessels, meaning that there is a period of time postoperatively during which the patient remains at risk of stroke.

Choosing the type of approach remains a controversial subject, often the outcome of institutional preference and surgeon comfort. There is a dearth of evidence-based data to drive decision making. Overall, children are predominantly treated with indirect approaches, about 75% indirect: 25% direct, with American Heart Association Guidelines supporting the use of indirect approaches in younger children.[10,13] Adults, on the other hand, more commonly undergo STA–MCA bypass.[14,15]

37.5 Surgical Procedure

Any moyamoya surgery requires meticulous perioperative attention to detail. While each surgeon will individualize his or her approach, there are general principles that can be applied to most cases. These principles will be illustrated through descriptions of one of the most common moyamoya operations, pial synangiosis (▶ Fig. 37.1**, Video 37.1**).

37.5.1 Preoperative (Day 1)

Ensure imaging is concordant with diagnosis, confirm side(s) and arrange anesthesia evaluation. Admit to hospital the night before surgery for intravenous hydration (usually 1–1.5 times baseline rate if otherwise healthy) and—if on aspirin—administer dose the day before (but not the day of) surgery.

37.5.2 Preoperative (Morning of Surgery)

Consider electroencephalography monitoring, place array if patient able to tolerate leads (ensuring leads are not in planned surgical field). Discuss plan with anesthesiologist, continue intravenous fluids, avoid stress of hypotension with induction, and administer antibiotics.

37.5.3 Equipment

Microscope, Doppler ultrasound (including micro-Doppler if direct bypass planned), fine curved snap, bipolar, craniotome, microdissection kit (jeweler's forceps, microscissors, microneedle driver, tying instruments, arachnoid knife), heparin saline, rubber dam, 10-nylon sutures (BV needle), and Gelfoam.

37.5.4 Positioning

Once anesthetized, place Mayfield with pins along sagittal axis (one for forehead and two near inion). Position supine, head turned opposite to side to be treated, with shoulder roll to reduce excessive rotation of the neck. Elevate the head and ensure that the operative field is flat, parallel to the floor (▶ Fig. 37.2).

37.5.5 Map the STA

Typically the parietal branch of the STA will be the donor. Dissection often extends from the root of the zygoma in front of the external auditory meatus superiorly and posteriorly about 10 to 15 cm (depending on the age and size of the patient). A small strip of hair is clipped and the course of the vessel can be mapped out using the Doppler and marking with a surgical pen or scratching the skin with a needle. Care should be taken to ensure that the artery is being mapped and not the vein. Do *not* inject the field with local anesthetic to avoid the risk of vessel injury.

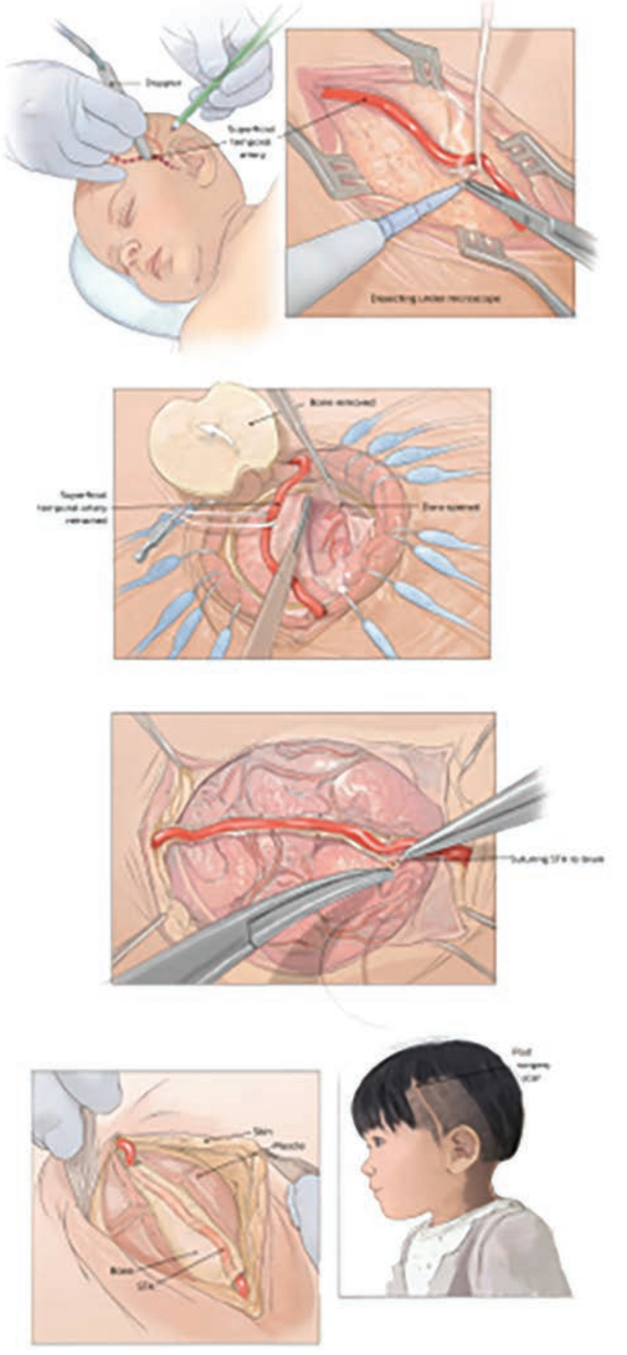
Doppler
Superficial temporal artery
Dissecting under microscope
Bone removed
Superficial temporal artery retracted
Dura opened
Suturing STA to brain
Skin
Muscle
Bone
STA
Post surgery scar

Fig. 37.1 Overview of indirect procedure, pial synangiosis.

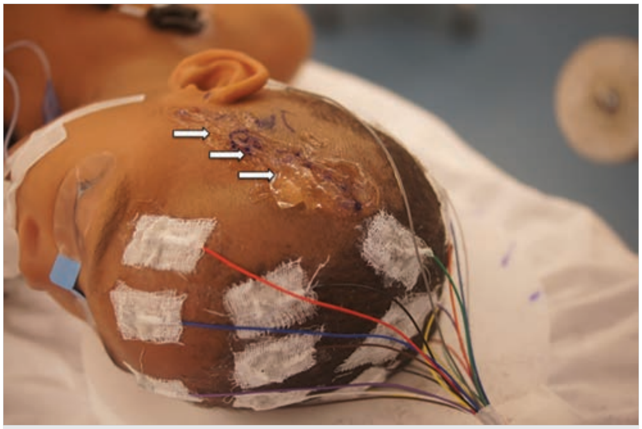

Fig. 37.2 The young patient's head rests in a donut, with EEG leads placed in an array avoiding the planned operative field. The course of the parietal branch of the superficial temporal artery is mapped out with purple marker and scratches under a thick layer of ultrasound gel. Note the flat position of the operative field and the elevation of the head.

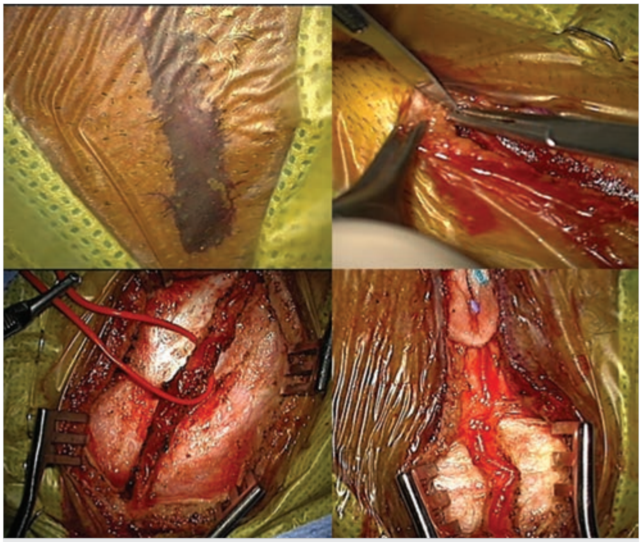

Fig. 37.3 The vessel dissection is outlined in clockwise rotation, starting in the upper left. Under the microscope, the distal end is the starting point. The next image in the upper right demonstrates the curved snap dissecting the vessel, with a number 15 blade opening the skin above the superficial temporal artery (STA). The lower right image depicts the creation of a vessel cuff, using a self-retaining retractor as one dissects in short, equal lengths on either side of the vessel. The lower left image is shows the use of a vessel loop under the STA, enabling the surgeon to elevate the vessel off the temporalis muscle.

37.5.6 Dissect the STA

The initial operative step is to dissect the length of the STA. One practice is to start at the distal end, near the vertex of the head, in order to proceed in a natural direction for the surgeon, away from oneself. This is often done under the microscope and begins with a sharp opening in the dermis (usually with a number 15 blade), followed by gentle subcutaneous dissection with a curved snap (▶ Fig. 37.3). The snap is then held closed over the vessel and the assistant can then incise the skin, exposing a length of STA. This process is until the length of the vessel is exposed. Following this, side branches are cauterized with the bipolar and sharply divided. Lastly, a vessel loop is passed under the STA and the electrocautery is used to dissect the vessel off of the temporalis muscle. It is helpful to leave a generous cuff of tissue around the vessel, which encourages blood vessel sprouting.

37.5.7 Craniotomy

At this point, the microscope is removed and a plane between the galea and temporalis is defined, extending both anteriorly and posteriorly from the STA. The temporalis is then divided in quadrants, one axis along the STA and the other perpendicular to it. This affords a wide opening for the craniotomy. Retractors are placed and a craniotomy is performed. Care must be taken to protect the vessel. Often two burr holes are placed—one at the apex and one at the base of the vessel. After stripping the dura from the bone with a Penfield #3, the foot plate is used with the craniotome to turn a large circular flap (▶ Fig. 37.4).

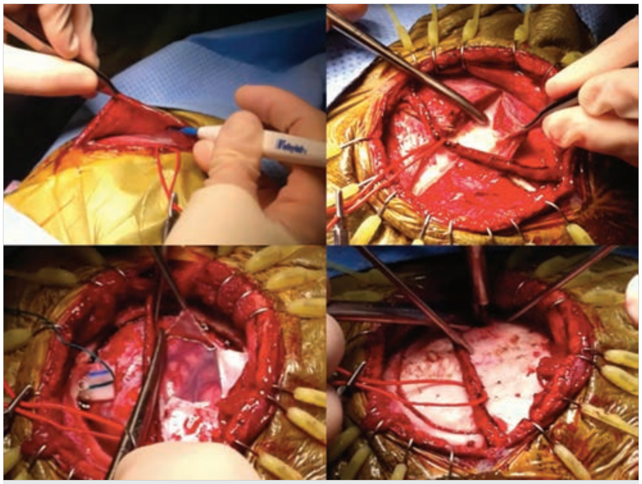

Fig. 37.4 The muscle dissection, craniotomy and dural opening are depicted in clockwise rotation. The upper left image shows the creation of a plane between the galea and temporalis muscle. The upper right depicts how the muscle is divided into quadrants and the lower right shows the burr holes and footplate turning a craniotomy flap. The lower left is an image of the dural opening, with wedge-shaped leaflets.

37.5.8 Dural and Arachnoid Opening

It is critical to assess preoperative films to identify any spontaneous transdural collaterals and avoid injuring them at opening. The dura acts as an additional source of blood supply, regardless of technique used, so it is important to minimize dural cautery (especially if there are known transdural collaterals). Bringing the operating microscope back into the field, an arachnoid knife, jeweler's forceps, and microscissors are used to widely open the arachnoid. Care must be taken not to injure the brain or vessels; any bleeding can often be controlled with brief gentle tamponade rather than cautery (▶ Fig. 37.5).

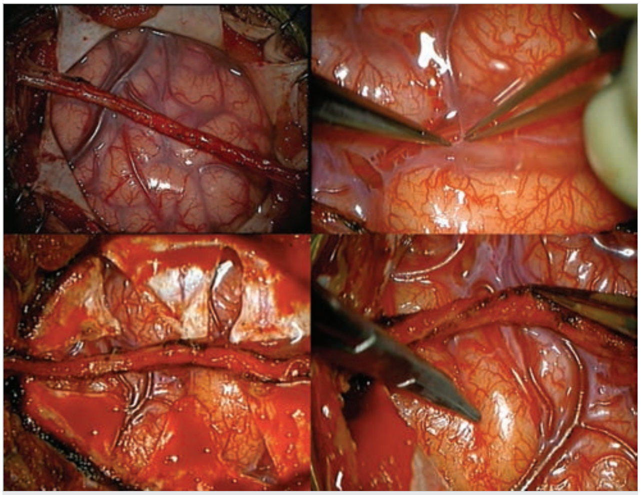

Fig. 37.5 Clockwise from upper left, the intradural steps of the synangiosis. First, with the dura open, the field is inspected for areas to open the arachnoid. In the upper right, the arachnoid is opened with jeweler's forceps, using the natural plane adjacent to a cortical artery. Below that is the synangiosis, using a 10–0 nylon. Note the depth of the needle and the anticipated area of the superficial temporal artery suture on the underside of the vessel. Lastly, the lower left image shows the conclusion of the synangiosis, with replacement of the dural flaps without any dural sutures. The Gelfoam will be placed on top of this area to cover it completely.

37.5.9 Synangiosis

At this stage, the goal is to affix the STA to the pial surface in order to maximize contact of the donor vessel with the brain surface to promote the ingrowth of new vessels. This can be achieved through the placement of multiple sutures from the cuff of the vessel to the pial surface. The use of 10–0 nylon with BV 75 micron needles works well, with care taken to ensure that the needle placement is shallow enough to avoid deep hemorrhage and that the entry and exit points do not traverse or pierce a dilated cortical vessel (▶ Fig. 37.5).

37.5.10 Closure

Once the synangiosis or bypass is completed, the microscope is removed, the dural leaflets are replaced loosely on the brain surface (without suturing and after inspecting for bleeding), and a large piece of Gelfoam is placed over the craniotomy site. The bone flap is then carefully replaced (avoiding compression or injury to the STA), often with rigid fixation. The muscle is then closed superiorly to inferiorly using absorbable sutures (leaving the horizontal plane open to avoid compressing the vessel). Galea and skin are closed and the wound is dressed (▶ Fig. 37.6).

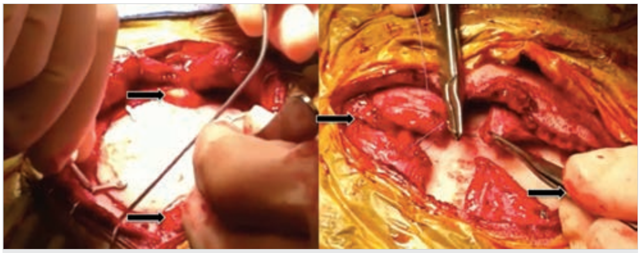

Fig. 37.6 Closure consists of replacing the bone flap with low profile plates on the left image (entry and exit points of the superficial temporal artery marked with arrows). The muscle is closed in the plane perpendicular to the vessel (right image, vessel entry and exit marked with arrows).

37.5.11 Intraoperative Problems

While specific technical problem areas have been addressed concordant with the stage of the operation, there are general problems that can occur at any time during a moyamoya procedure. Electroencephalography slowing can herald reduced cerebral blood flow (possibly from spasm or blood pressure changes) and bolus administration of propofol may serve to reduce metabolic demand of the brain and thereby provide a neuroprotective effect. Bleeding maybe particularly troublesome and maybe more pronounced if aspirin is used. Meticulous hemostasis is crucial, although "over-cautery" will only serve to deprive the brain from potential additional sources of blood supply. Brain swelling (unrelated to direct bypass) can create a cycle of reduced venous outflow—feeding more swelling. Elevation of the head of the bed, opening of arachnoid to drain cerebrospinal fluid, and increased sedation are all tools to help. Hyperventilation and driving down pCO_2 should be avoided in moyamoya patients, as this may precipitate vasoconstriction and stroke in a brain with a tenuous blood supply.

37.6 Postoperative Care, Complications, and Avoidance

Immediate postoperative care should be administered in the intensive care unit, with the goals of avoiding hypotension and hypocarbia. Generally patients are extubated, awake, and have an arterial line (for blood pressure management) and a bladder catheter (for monitoring volume status). Antibiotics are used for 24 hours. Aspirin is administered on postoperative day 1. Antiepileptics are not routinely prescribed. Intravenous fluids are run at 1 to 1.5 times maintenance and slowly decreased as the ability to take oral fluids recovers. Pain control is important and frequent neurological examinations are critical to detect any changes in exam. Any moyamoya patient is at risk of stroke in the perioperative period.

The greatest risks include stroke, hemorrhage, and problems with wound healing (infection, cerebrospinal fluid leak) and these risks are minimized through a combination of clear communication with team members and careful adherence to operative protocols throughout the hospital stay.[8,16] Hypotension and hyperventilation (causing reflexive cerebral vasoconstriction) should be avoided. Postoperatively, frequent neurological examinations are critical, especially the first day after operation. Changes should be reported and assessed. Direct bypass patients should be watched for hyperperfusion syndrome (sometimes treated with careful reductions in blood pressure), while ischemic symptoms may require increased blood pressure or administration of neuroprotective agents (such as propofol).

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38 Positional Compression of the Vertebral Arteries

David W. Newell and Dennis A. Velez

Abstract

Transient vertebrobasilar insufficiency (VBI) caused by head turning, also known as head turning syncope, is increasingly being recognized as a potentially incapacitating and debilitating condition. It is important to recognize the classic symptoms and understand the diagnostic workup that are important to establish an anatomic lesion responsible for clinical symptoms. Transient compression of the vertebral artery (VA) can also cause injury, dissection, occlusion, and transient embolic events and stroke. Fortunately, VBI can be successfully treated in many cases by surgical intervention. Excellent results depend on making a correct diagnosis and defining the site of VA compression. We discuss in this chapter, normal and abnormal anatomy of the vertebral basilar circulation as well as anatomical variants that in combination with transient vascular obstruction can lead to brain ischemia with neck movements, particularly head turning.

Keywords: vertebrobasilar ischemia, bow hunters stroke, head turn syncope, vertebral artery compression, posterior circulation ischemia

38.1 Normal Anatomy, Sites of Compression, and Symptoms

The vertebral artery (VA) is divided into four segments, termed V1–V4. The V1 or ostial segment arises from the subclavian artery and enters the transverse foramen of the C6 vertebra in most cases. Compression of the artery in this location may be due to fibrous bands or the anterior scalene muscle or tethering between the arterial origin and the transverse foramen of the C6. The V2 or transverse segment extends from the entry into the vertebral foramen, to the transverse foramen of the axis. Positional compression of the VA is the most common at the C1–C2 region due to the extreme mobility of this segment and at the subaxial cervical spine, which can occur from osteophytes at the transverse foramina, hypertrophy of the uncinate processes, or facets or cervical disc rupture. The V2 segment is surrounded by a venous plexus. This segment courses vertically until the C3 vertebra exits this vertebra's transverse foramen, then bends horizontally and laterally along the base of the axis and then turns upward to enter the transverse foramen of the axis. The V3 or suboccipital segment begins at the transverse foramen of the axis and ends at the dura mater of the foramen magnum. The course of this segment is the most complex and is the most mobile segment during head turning and therefore is potentially most vulnerable to being stretched and compressed during head movement. The V4 segment refers to the segment of the VA between the entry through the dura and the end of the artery at the vertebrobasilar junction.

Symptoms of rotational vertebrobasilar insufficiency (VBI) include syncope or near syncope, dizziness, vertigo, visual disturbances, drop attacks and motor, and sensory and cranial nerve deficits that are induced when the head is turned to a particular side.[1,2,3,4] With true rotational VBI, these symptoms may resolve promptly once the head is returned to the neutral position. Symptoms that persist or occur after the head has returned to neutral have been called "bow hunter's stroke" after the description of a lateral medullary stroke occurring in a patient with posterior circulation infarction after target practice.[5] There is also a syndrome called beauty parlor stroke that has been described in patients after having their hair washed with their head in extreme extension.[6] These conditions most likely arise from arterial injury and dissection and thromboembolic phenomenon. It is also likely that chiropractic manipulation can cause injury to the VA on rare occasions which may manifest as immediate or delayed thromboembolic events.[6]

Extrinsic compression of the VA by osteophytes or at the highly mobile C1–C2 segment during head rotation has been documented in cadaveric specimens and in patients (▶ Fig. 38.1).[4] Unilateral complete VA flow obstruction can occur in healthy people with head turning but VBI symptoms usually don't occur because the contralateral VA may provide the necessary compensation to perfuse the basilar artery during transient compression of either artery. Most patients with positional VBI have one patent VA and a contralateral VA stenosis/occlusion due to congenital hypoplasia, atherosclerosis, or other causes or the VA may not connect to the basilar artery due to an absent segment between the posterior inferior cerebellar artery and the VB junction. If the posterior communicating arteries are robust and can provide adequate collateral circulation, then even temporary occlusion of a single isolated VA may not cause ischemic symptoms. A model of the cervical spine and VA in a (left) neutral position and when the head is turned to the right showing that the VA is stretched by the forward projection of the C1 transverse foramen as the head (▶ Fig. 38.1).

38.2 Diagnosis

Our experience suggests that the specific clinical symptoms in positional VB ischemia are very consistent and can predict which patients will have true transient arterial obstruction and neurological symptoms from blood flow reduction.[2] Symptoms are most commonly associated with head turning, but may also be associated with head flexion or extension. Patients with the true disorder, usually report that they can reliably reproduce the symptoms by moving their head into a certain position and holding it there for a brief period of time (usually about 5–7 seconds) before the symptoms begin. Normally, patients will return their head to a neutral position as soon as symptoms begin, in order to avoid more severe symptoms or syncope. Symptoms include generalized weakness, dizziness, vertigo, diplopia, disorientation, and even altered level of consciousness.

Patients who report milder symptoms when they turn their head in different directions or inconsistently with flexion or extension or while looking up and cannot reproduce the symptoms in an office setting usually don't have VB flow obstruction or ischemia and often have vestibular disorders or nonspecific entities. We reported two patients with adolescent stretch syncope

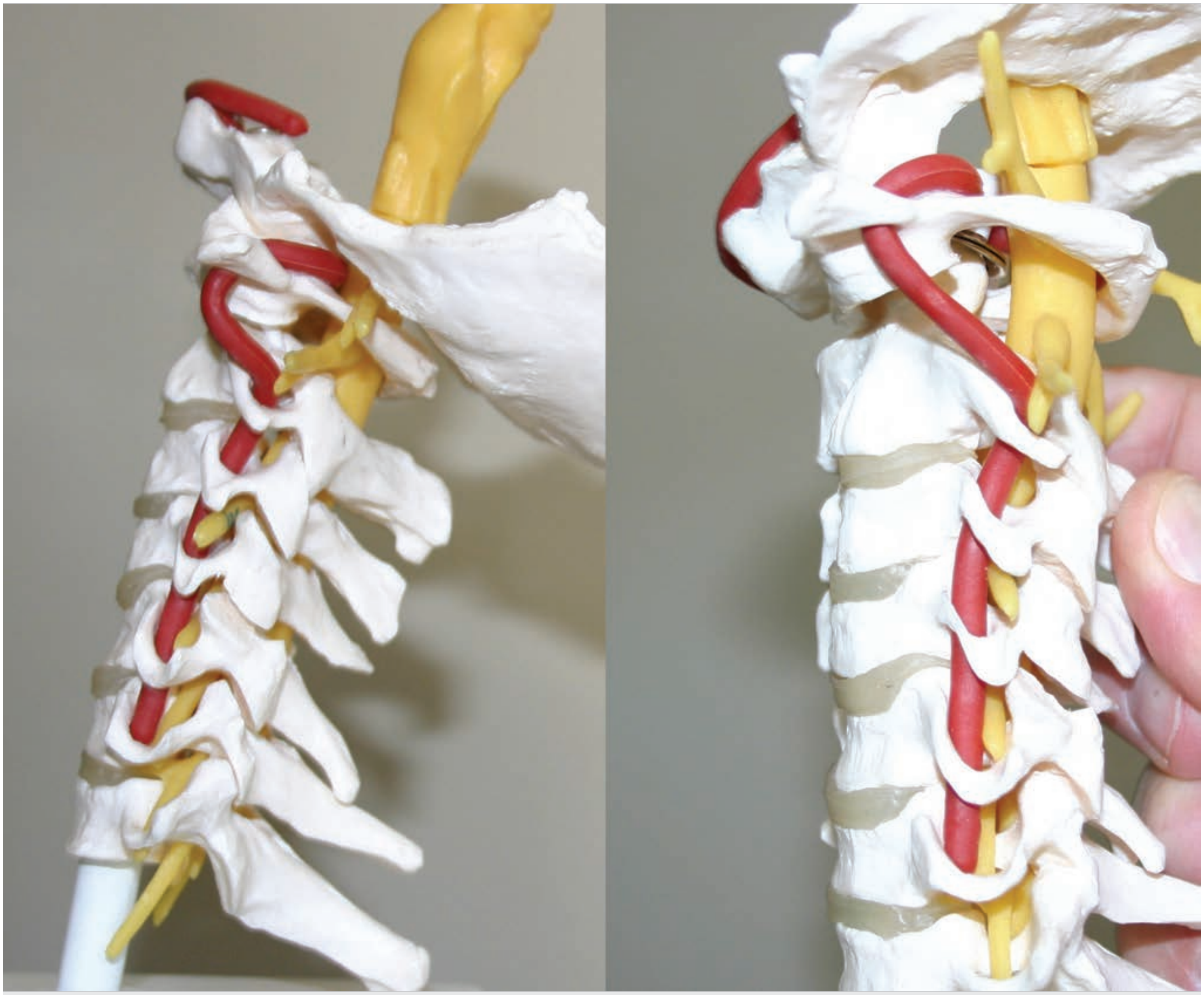

Fig. 38.1 (Left) Illustration of a plastic model that shows the vertebral artery passing through the transverse foramen of C1 in the neutral position and also (right) with the head turned to opposite side illustrating the mechanism of stretching of the vertebral artery at this location between the exit from the bone at C2 and the posterior part of the ring of the foramen at C1. This is the rationale for removing the posterior portion of the transverse foramen of C1 to prevent the stretching and occlusion of the lumen of the artery.

and were able to document a transient decrease in cerebral blood flow that was most likely due to a combination of stretching of the first or ostial segment of the VA and Valsalva maneuver.[3] Our patients experienced symptoms only with extreme postural positions and therefore were not advised to have surgical treatment.

Another syndrome characterized by presyncopal symptoms to be distinguished from VBI is Eagle's syndrome or variations of it. Eagle's syndrome describes odynophagia and neck and face pain secondary to compression of the carotid artery and other structures in the upper neck and pharynx by an abnormally large styloid process.[7] Other neurological conditions and cardiopulmonary status of the patient are also important to assess as causes of syncope/presyncope/dizziness prior to conducting more invasive, definitive diagnostic studies. Transcranial Doppler (TCD) ultrasonography, dynamic cerebral angiography, including the cervical VA and computed tomography angiography (CTA) of the neck and head are the key diagnostic tools when suspecting positional VBI.

TCD is an inexpensive, noninvasive first diagnostic tool. Dynamic TCD allows monitoring of hemodynamic changes with the patient's symptoms. In patients with positional VBI, the blood flow velocity in the posterior cerebral arteries decreases with head rotation to at least 50% of the patient's baseline with the head in the neutral position. Moreover, a reactive hyperemic response of at least 10% above the baseline occurs when the patient resumes the neutral position and the symptoms disappear.[2] These findings are consistently reproducible in patients with true rotational VBI (▶ Fig. 38.2**, Video 38.1**). Monitoring is done with bilateral fixed probes, which minimizes motion artifacts and allows for simultaneous recordings of both posterior cerebral arteries. TCD can also be invaluable in the operating room to help confirm complete decompression of the VA by detecting normal posterior cerebral artery flow velocities while performing passive head turning maneuvers. This technique, however, is limited to patients who are supine and when their head is not fixed and it is safe to rotate their head around when they are intubated under

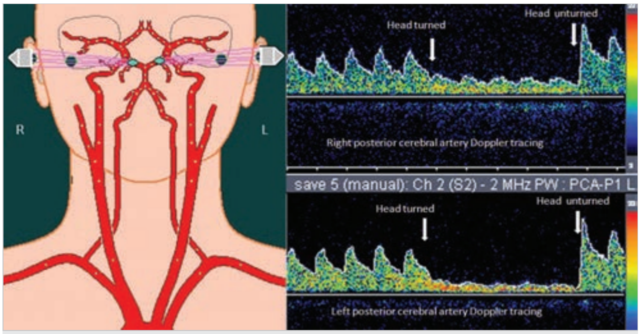
R
L
Head turned
Head unturned
Right posterior cerebral artery Doppler tracing
save 5 (manual): Ch 2 (S2) - 2 MHz PW : PCA-P1 L
Head turned
Head unturned
Left posterior cerebral artery Doppler tracing

Fig. 38.2 (Left) Illustration of a diagram of the simultaneous transcranial Doppler recording of both posterior cerebral arteries. (Right) Spectral and outline of the posterior cerebral artery velocity profile initially in a neutral position and then with head turned, with accompanying velocity drop in a symptomatic patient, followed by return of the head to a neutral position and a hyperemic response in the velocity profile, confirming that there was a period of cerebral ischemia.

general anesthetic. TCD is also valuable to document resolution of positional blood flow reductions, postoperatively.

Dynamic catheter angiography with the patient participating in head rotation and multiple views allows for precise identification of the site of VA compression and shows the detailed vascular anatomy. The entire course of both VA needs to be visualized and carefully studied to prevent misdiagnosis. CTA can also be done with the head in neutral and rotated positions; however, it is often of limited value if the patients cannot maintain the head position which produces the symptoms for more than a few seconds. It may give additional information about the site of bony compression and is also useful to document postoperative VA decompression.

38.3 Treatment Options

Advising the patient to limit head rotation is a treatment option. This can be advised in patients who only experience mild symptoms, symptoms in extreme head positions, or those who are not surgical candidates. Surgical treatment is indicated in patients with recurrent incapacitating symptoms or those with signs and/or neuroimaging (TCD, CT, MRI, and CTA) that demonstrates hemodynamically significant compression of the VA. The surgical procedure is dictated by the site of compression and angiographic and CT findings. In the subaxial spine, the most common pathology is hypertrophy of the uncovertebral joint producing compression and periarterial fibrosis of the VA. In these cases, the VA is compressed to the point of temporary arterial occlusion upon head turning. It is our experience that the transient compression and arterial narrowing is produced when the head is turned *toward* the side of the lesion. There are also reports of lateral disc herniation producing a similar degree of positional arterial narrowing and transient VBI symptoms.[8]

Transient VBI may be due to embolism or hemodynamic insufficiency. Chronic stretching and compression of the VA can lead to intimal injury, thrombus formation, embolization, and ischemic stroke. Medical treatment with antiplatelet and anticoagulation agents may be indicated in such cases. Endovascular treatment of positional stenosis of the VA with balloon angioplasty and stenting is seldom been reported but would be unlikely to be effective in such cases.

38.4 Operative Procedures

38.4.1 Anterior Approach to the Subaxial Spine

In the case of osteophytes arising from the uncinate process, an anterior approach with lateral retraction of the longus colli allows for exposure of the transverse process above and below the level of compression (▶ Fig. 38.3 **and** ▶ Fig. 38.4). General anesthesia is induced. The patient is positioned supine with the head in neutral position as for an anterior cervical discectomy. Similar instruments and retractors are all that are needed. Fluoroscopy is useful to localize the level. Decompression may be needed over several levels to completely free the VA, so a longitudinal incision may be preferable although certainly not necessary. The initial dissection is the same as for an anterior cervical discectomy and along the plane between the sternocleidomastoid and carotid sheath laterally and the strap muscles, trachea, and esophagus medially. It is on the side of the compressed VA and exposure has to extend laterally over the longus colli muscle. The muscle is dissected off the vertebral body and retracted laterally over the affected level as well as the level above and below. The transverse processes are identified and the anterior surfaces, which form the anterior walls of the transverse foramen, can be removed with Kerrison rongeurs. This is generally done under the operating microscope. At the level of compression, the uncinate process and associated osteophyte can be removed with a high-speed drill, protecting the VA in its sheath. Once this is done, the VA can be mobilized laterally and the medially located osteophytes positioned more posteriorly are drilled away. The venous plexus around the VA and the surrounding fibrotic adventitia are incised along the length of the exposed VA, which may include the level of compression as well as one above and below, so as to achieve full decompression.[4]

Intraoperative Doppler ultrasound can be performed before and after decompression to document a decrease in high-flow velocity at the site of compression. Closure is routine. An external cervical collar is not necessary after the surgery.

38.4.2 Posterior Approach to the Subaxial Spine

In rare cases the osteophyte arises from the facet joint such that an approach from posteriorly is indicated (▶ Fig. 38.5). The patient is put under general anesthesia and placed in the prone position. The head is fixed in pin fixation. Fluoroscopy is used to localize the correct level. A midline posterior incision allows access to the offending facet. Instrumentation for a cervical laminectomy is adequate. A unilateral subperiosteal dissection is performed. The exposure is more lateral than for a cervical laminectomy because the facet complex has to be exposed. This facet can then be drilled with a high-speed drill with the help of an operating microscope. Once the facet is removed, the VA will be exposed but contained in a fibrous sheath and a perivascular venous plexus. This must be opened

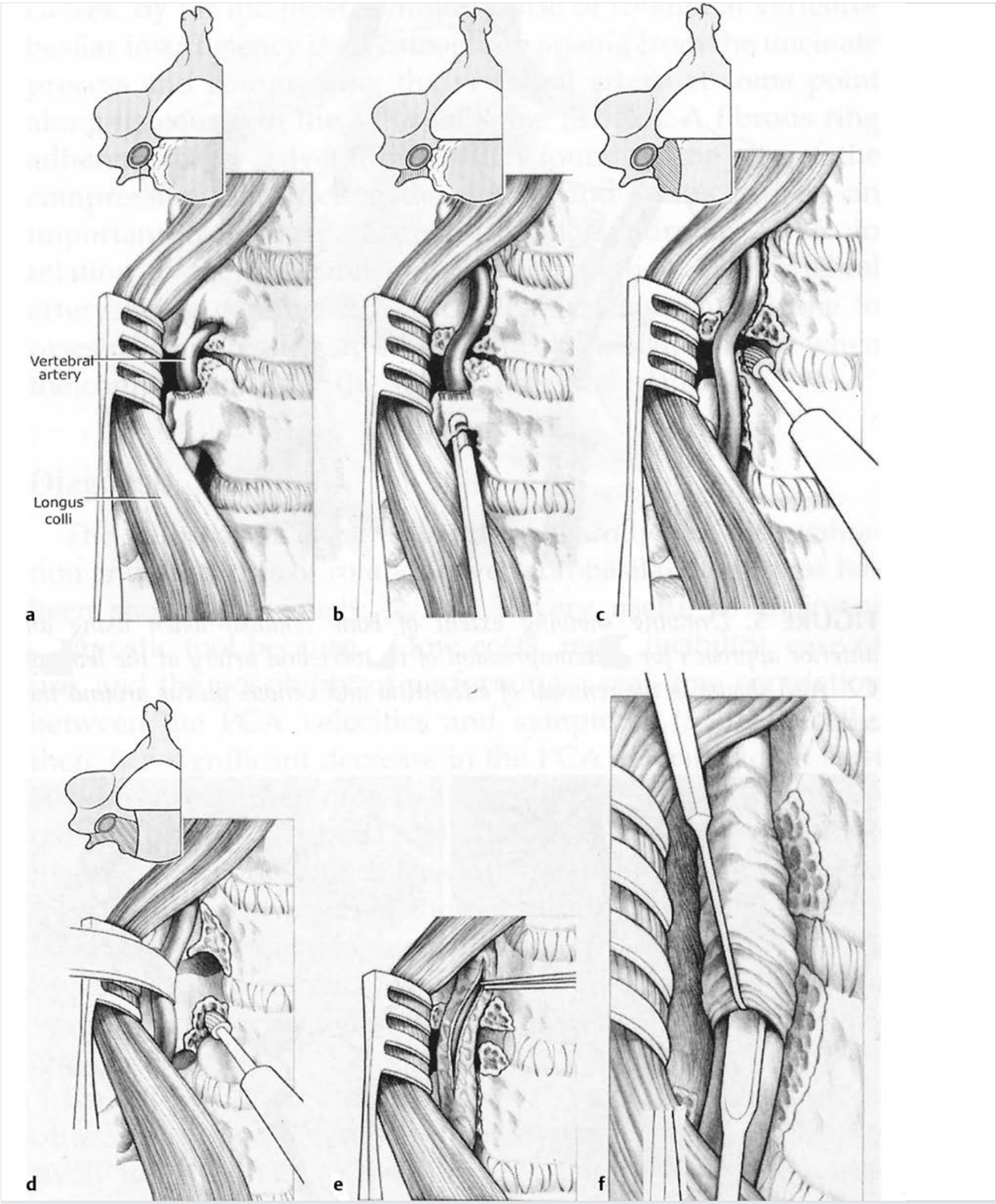

Fig. 38.3 Drawings of anterior approach to the vertebral artery (VA). **(a)** The longus colli muscle is retracted laterally to expose the ventral bony surface of the transverse foramen at multiple levels. **(b)** The anterior bony wall of the transverse foramen is removed with Kerrison punches. **(c)** The VA is then retracted laterally and a high-speed drill is used to remove the osteophytes of the hypertrophic uncovertebral joint that compress the artery. **(d)** Bone removal is completed. **(e)** The perivascular sheath and vertebral venous plexus are opened and coagulated to complete decompression of the VA. **(f)** The perivascular sheath can be opened after dissecting off the VA with a periosteal elevator and a hook.

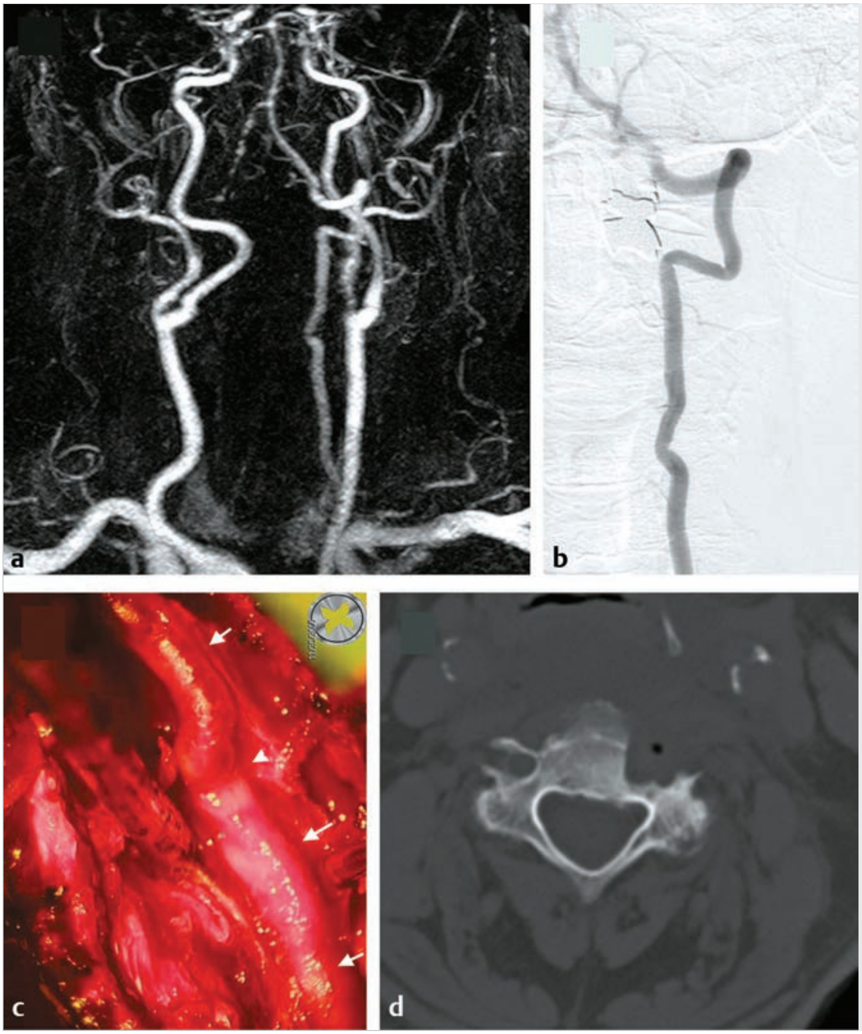

Fig. 38.4 A 63-year-old man presented with an episode of loss of consciousness when he extended his head to look up. He subsequently had a sensation of impending loss of consciousness whenever he extended his neck. **(a)** Preoperative magnetic resonance and **(b)** left vertebral artery (VA) angiography show kinking of the artery at the interspace between C3 and C4 vertebra. The right VA was occluded, the left posterior communicating artery was small, and the right was large but with a small right precommunicating posterior cerebral artery segment. An anterior decompression of the VA was performed. **(c)** An intraoperative photograph shows the decompressed VA *(arrows)*. The site of kinking is visible *(arrowhead)*. **(d)** Postoperative computed tomographic scan demonstrated decompression of the left VA. The patient was able to extend his neck after surgery and had no further symptoms.

to ensure the artery is decompressed. Intraoperative Doppler ultrasound can be used to confirm that the velocity of flow along the VA above, at, and below the stenosis is the same. Removal of one facet should not cause instability and spinal fusion is generally not necessary, nor is a cervical collar. The closure is routine.

38.4.3 Posterior Approach to the Atlantoaxial Level

Three approaches have been described for decompression of the VA at the atlantoaxial level: anterior decompression at the transverse foramen of the atlas, posterior decompression at the transverse foramen of the atlas, and posterior fusion of the atlas (C1) and axis (C2, ▶ Fig. 38.6).[4,9] For posterior decompression of the atlas, which is the most commonly performed procedure, the patient is put under general anesthesia and placed in the prone position and the head is secured in pin fixation. A midline suboccipital incision is made and the posterior arch of the atlas and axis are exposed on the affected side. The exposure has to be carried laterally over the transverse process of the atlas and the lateral mass of the axis. The VA travels over the posterior arch of the atlas laterally in a palpable groove, between the atlas and foramen magnum and care should be taken not to injure it at the site. It is surrounded in a venous plexus, the exposure of which alerts the surgeon to the underlying artery. The plexus bleeds profusely and is controlled with coagulation, division and topical hemostatic agents such as Gelfoam (Pfizer, Inc., New York, NY) or similar agents containing thrombin. The artery is followed laterally and inferiorly to the transverse foramen of the atlas, which is unroofed with a high-speed drill, Kerrison punches and Lempert rongeurs. It is also important to mobilize the artery and make sure it is not tethered by soft tissue adhesions. Once the site of compression has been identified and relieved, intraoperative Doppler ultrasound can be used to confirm decompression. The wound is closed.

38.4.4 Other Operative Treatment Options

There are other operative treatment options that have been described for treating positional obstruction of the VA including posterior fusion of the C1–C2 joint with or without direct decompression of the VA. In the authors opinion, fusion is not required if there are no other pathological conditions which accompany the positional obstruction which require fusion, either at C1–C2 or in the subaxial spine. If, however, there is bilateral cervical nerve root and or spinal cord compression

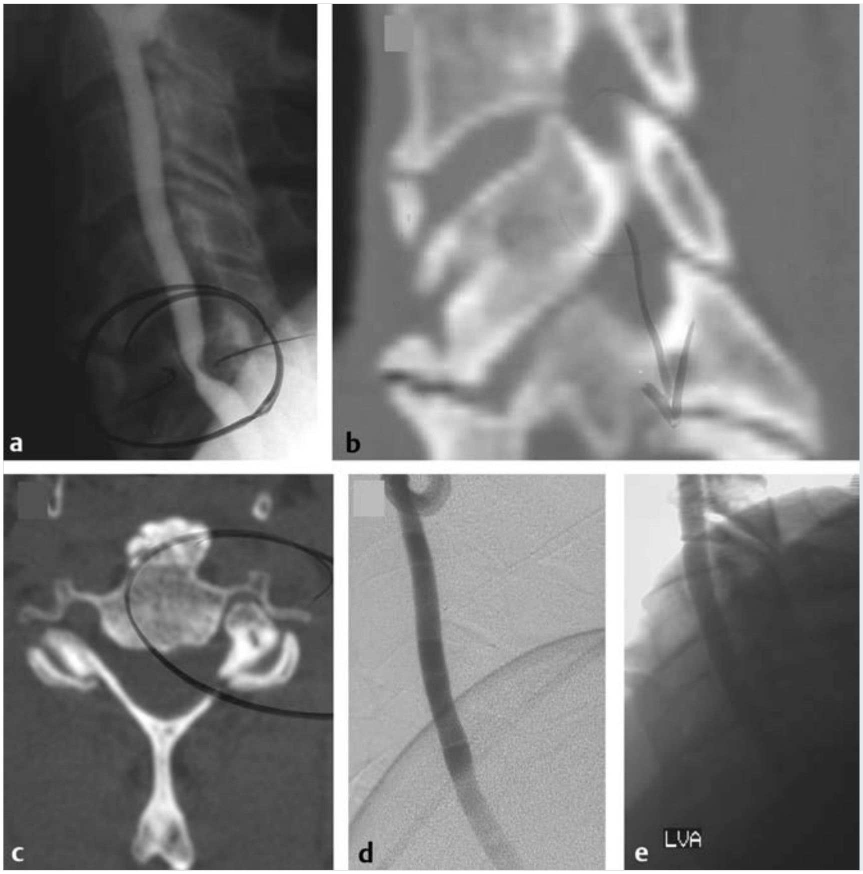

Fig. 38.5 A 69-year-old male had a 15-year history of intermittent vertigo precipitated by moving from the supine to the upright or the upright to the supine position. He then had a transient episode where he extended his head and developed sudden onset of vertigo, dysarthria, horizontal diplopia, and tetraparesis. **(a)** Lateral left vertebral angiography with the head in the neutral position showed severe focal stenosis of the left vertebral artery (VA) at the C6 vertebra. The right VA had a stenosis in its fourth segment. **(b)** Sagittal computed tomographic (CT) scan reconstruction. **(c)** Axial CT scan of the cervical spine at the facet complex of the fifth and sixth vertebrae showed an osteophyte from the superior facet of the C6 vertebra that narrowed the transverse foramen and VA. This was treated by posterior decompression with removal of the facet complex and osteophyte and exposure of the VA. **(d)** Postoperative subtraction and **(e)** un-subtracted angiography confirmed that the VA was of normal caliber. The patient's symptoms resolved.

which require a more extensive decompression, then the addition of fusion is often required.

38.5 Postoperative Management Including Possible Complications

The patient should be monitored postoperatively in an intensive care or step-down unit. We consider early postoperative treatment with acetylsalicylic acid. Otherwise, no specific early measures are necessary. Discharge home is within a few days and guided by the usual considerations. Postoperative pain usually limits neck movement for days so that imaging can be delayed for weeks. Once the patient can move normally, either or both Doppler studies and a CTA with the head in the neutral and rotated or extended positions can be obtained to determine if the decompression is adequate and the symptoms have resolved.

The complications of surgery can include hematoma, VA injury, cervical nerve root injury, and failure to adequately decompress the VA. If the VA was lacerated at surgery, temporary trapping and repair would be mandatory because in most cases this would be the only vascular supply to the posterior fossa. Failure to decompress is possible if there is inadequate release of the fibrous tissue around the artery, which can remain as a constricting band even after the bone is removed.

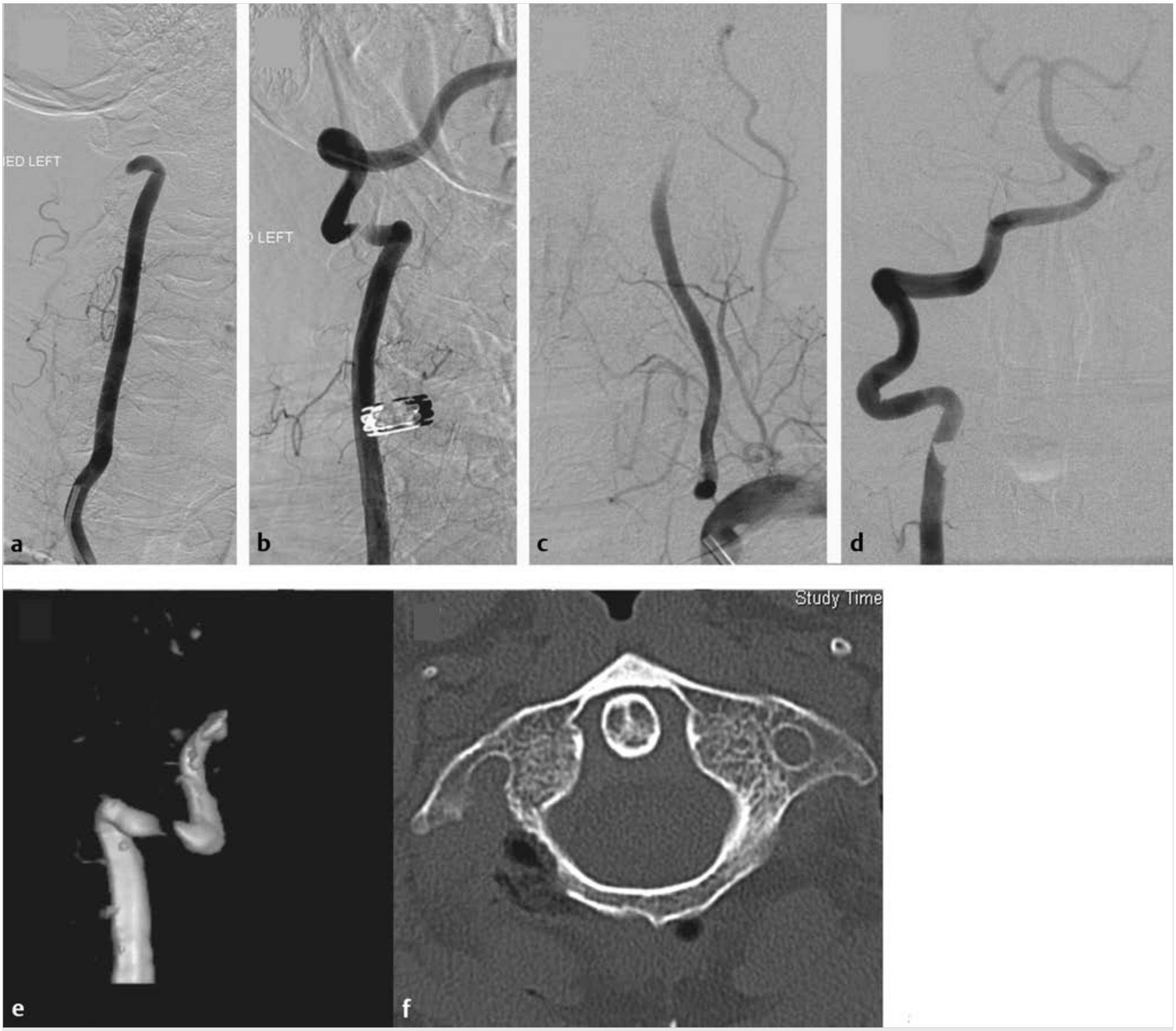

Fig. 38.6 A 54-year-old male presented with dizziness and nausea when turning his head to the left. He was found to have an occluded left vertebral artery (VA) and positional obstruction of the right VA at C1–C2 with head turning to the left. **(a)** Right VA injections with the head turned to the left show occlusion and **(b)** stenosis at the right VA with marked and moderate head turning, respectively. **(c)** The left VA is occluded at the mid-cervical region. **(d)** With the head in the neutral position, the right VA robustly fills the posterior circulation. **(e)** A computed tomographic (CT) angiogram shows the area of stenosis of the right VA with head turning. **(f)** Postoperative CT scan after posterior decompression shows the transverse foramen of the atlas is opened.

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39 Minimally Invasive Approaches for Spontaneous Intracerebral Hemorrhage

Jennifer Kosty, Norberto Andaluz, Chiraz Chaalala, and Mario Zuccarello

Abstract

Minimally invasive surgical (MIS) evacuation of intracerebral hemorrhage effectively reduces clot burden with minimal disruption of viable tissue. Recent literature suggests that MIS evacuation may lead to improved functional outcomes compared with conservative management or conventional craniotomy. The surgical technique consists of inserting a catheter into the clot under stereotactic guidance with subsequent aspiration of blood products. With the catheter left in place, its position is then confirmed with a postoperative computed tomography. Episodic administration of recombinant tissue plasminogen activator into the residual hematoma with subsequent drainage is performed for up to 72 hours, or until the remaining clot is reduced by 80% or to 10 to 15 mL as measured by the ABC/2 method. This procedure reduces the volume of clot and perihematomal edema. Future studies are needed to assess the effectiveness of this procedure for improving patient-oriented outcomes.

Keywords: intracerebral hemorrhage, stroke, minimally invasive, stereotactic surgery

39.1 Spontaneous Intracerebral Hemorrhage

Spontaneous intracerebral hemorrhage (ICH) accounts for nearly 2 million strokes worldwide per year with a mortality rate approaching 50% at 30 days and only 25% of survivors achieving functional independence.[1,2] Recent literature suggests that diffusion tensor imaging, which defines the location and integrity of a white matter tract maybe used to predict both motor and functional in patients with ICH.[3,4] Risk factors for ICH include hypertension, older age, male sex, African American and Japanese populations, alcohol or street drug abuse, liver dysfunction, vasculopathy, and anticoagulation.

Spontaneous ICH occurs most commonly in the putamen followed by the subcortical white matter, putamen, thalamus, cerebellum, and pons.[5] Hemorrhage in the basal ganglia is thought to be due to the rupture of Charcot–Bouchard microaneurysms that form in the small perforating arteries as a result of hypertension-induced degenerative changes in the vessel wall. A lobar hemorrhage accounts for up to 50% of ICHs and is more likely to be associated with an underlying cause such as amyloid angiopathy, anticoagulation, vascular malformations, or tumors. The proportion of ICHs secondary to warfarin and other anticoagulants, now estimated at 20%, maybe increasing due to increasing use of anticoagulants.[6,7]

39.2 History of Minimally Invasive Surgery for Intracerebral Hematoma

The role of surgical evacuation in the management of ICH is controversial. Cushing probably performed the first surgical aspiration of an ICH in 1903. This initiated an ongoing debate and a study of the safety and efficacy of this practice that has persisted over the ensuing 60 years. In the first randomized controlled trial in neurosurgery by McKissock and colleagues in 1961, patients randomized to surgical intervention fared worse than those who received conservative management.[8] Multiple small subsequent trials with conflicting results prompted the International Surgical Trial in Intracerebral Haemorrhage (STICH) to date the largest randomized controlled trial in the field.[9] Approximately 1,000 patients were randomized to surgical evacuation within 24 hours or best medical management. Rates of mortality and favorable outcome were comparable in both groups, but a 26% crossover rate to surgical intervention was observed. Subgroup analysis demonstrated significantly improved outcomes in those patients with ICHs extending to within 1 cm of the cortical surface who underwent surgery. As a follow-up, STICH II randomized 600 patients with lobar ICH to surgical evacuation versus medical management.[10] A nonsignificant trend toward improved outcome and decreased mortality was reported. In both studies, open craniotomy was the primary surgical technique.

Given the failures of STICH I and STICH II, the role of MIS evacuation has gained increasing attention. MIS techniques decrease ICH-related mass effect and reduce the burden of potentially toxic blood breakdown products while minimally disturbing viable brain. MIS techniques began in 1970s, with the first widely noted series by Backlund describing the use of an Archimedes' screw-type device.[11] Larger series published in the 1980s, particularly in Japan, reported that computed tomography (CT)-guided aspiration was associated with improved outcomes compared with conventional craniotomy.[12] Others reported success with endoscope-guided techniques.[13] After Niizuma and Suzuki first described the application of thrombolytics to assist in the evacuation of ICH, multiple groups have described the safety and efficacy of this practice.[14,15] In a 2011 study comparing conventional craniotomy with urokinase-assisted MIS evacuation of ICH in 122 randomized patients, Zhou et al found higher functional outcome scores in the MIS group.[16] Others have reported similar results in large, nonrandomized series.[17] Given the promise of MIS evacuation of ICH, the multicenter Minimally Invasive Surgery and recombinant Tissue plasminogen activator (rt-PA) in ICH Evacuation (MISTIE) study was developed and followed by MISTIE II, the safety and dosing phase was recently completed. Ninety-six patients were randomized to MIS plus rt-PA versus standard medical care.[18] There was no difference in 30 day mortality, symptomatic bleeding, or brain bacterial infections. MISTIE III is currently underway to evaluate the clinical efficacy and safety of this intervention.

39.3 Patient Selection

The treatment of spontaneous ICH depends on the patient's neurological and medical condition and the site and size of the hemorrhage. Location is a key factor—surgery plays a prominent role in management of cerebellar hemorrhage, it is controversial for deep basal ganglia hemorrhages, and it is generally contraindicated for brainstem locations.

We recommend MIS evacuation of deep or superficial ICHs in patients with a Glasgow coma score ≥ 8 with no underlying cause and with or without intraventricular hemorrhage. These procedures can be performed under local anesthesia, thus resulting in shorter operative times. Surgical removal is effective in patients with large lobar hemorrhages ($> 50\,\text{cm}^3$) that show transtentorial herniation. However, patients with supratentorial ICHs that extend into the brainstem are not good surgical candidates.

39.4 Preoperative Preparation

If the underlying cause suspected for the ICH is other than chronic systemic hypertension, then the etiology of the bleeding should be investigated. This includes complete blood count with platelet count, bleeding time, prothrombin time, partial thromboplastin time, liver function, and kidney function. Imaging studies should include magnetic resonance imaging (MRI), CT, CT angiography, and in some cases a catheter digital subtraction cerebral angiogram.

Any coagulopathy should be corrected preoperatively. Antiepileptics are indicated in patients who have had seizures, and may be used as prophylaxis. Fever and hyperglycemia should be treated. Mean arterial pressure should be kept below 110 mm Hg and systolic blood pressure below 160 mm Hg. In patients with known hypertension, blood pressure should be reduced 15 to 20% below baseline blood pressure. Intracranial pressure monitoring is used in patients with a Glasgow coma score of 8 or less and in those with hydrocephalus and/or intraventricular hemorrhage suspected to have elevated intracranial pressures. A thin-slice CT or MRI scan of the brain is obtained in preparation for frame-based or frameless stereotactic hematoma evacuation.

39.5 Operative Procedure

The procedure can be performed under general or local anesthesia. The patient's head is fixed in a stereotactic frame for frame-based stereotaxy or in a Mayfield head holder (Integra LifeSciences Corp., Plainsboro, NJ) for frameless stereotaxy. Positioning of the patient is based on the location of the hematoma. For frameless stereotactic procedures, patient registration is performed in standard fashion, using a six-point fiducial system or surface matching registration, according to the image guidance system used. A surgical trajectory with a final catheter placement along the major axis of the hematoma is planned to ensure a higher efficacy with the procedure, lower residual volumes, and lower rt-PA dosage. Preparation and draping are performed in a sterile manner.

39.5.1 Incision

A 2.5-cm incision is made over the affected area or in the frontal precoronal region in patients with deep ICH. A large burr hole is cut just posterior to the thickest part of the hematoma. The dura is incised (1 cm) in a cruciform fashion.

39.5.2 Hematoma Evacuation

A 14 French cannula (standardized introducer/peel-away sheath for endoscopy, Medtronic 14 F, 4.7 mm, Medtronic, Minneapolis, MN) is introduced with a single pass into the central core (two thirds of overall hematoma diameter) of the hematoma (▶ Fig. 39.1**a**). After removal of its inner portion, the cannula tself will remain within the intracerebral hematoma **(**▶ Fig. 39.1**b**). Using a 10-mL syringe, the surgeon aspirates the hematoma until the fluid component of the clot is no longer noted in the aspirate (▶ Fig. 39.1**c**). Documentation of the amount of aspirate is important in deciding when to stop the procedure.

A soft catheter, part of an external drainage ventricular set (Codman, Raynham, MA), is passed through the cannula into the residual hematoma (▶ Fig. 39.1**d**). As the cannula is removed, the surgeon ensures that the soft ventricular catheter remains within the residual hematoma (▶ Fig. 39.1**e**). The catheter is tunneled away from the incision and fixed to the skin. The incision is closed in layers in a standard fashion. The catheter is connected to a three-way stopcock and then to a closed drainage bag system (Codman ventricular catheter kit, Codman) (▶ Fig. 39.1**f**).

39.6 Postoperative Management Including Possible Complications

The drainage system remains open to drainage at zero pressure for 3 hours. A CT scan is performed 3 hours after surgery to ensure that the catheter is optimally positioned and to rule out rebleeding (▶ Fig. 39.2**a,b**). If thrombolytic agents are used, the first dose is given 3 or more hours after surgical placement of the catheter once CT has confirmed the size of residual clot is stable; thereafter, administration is repeated two to three times daily. Discontinuing the administration of thrombolytic agents is recommended after 72 hours of treatment or when the clot volume has decreased to about 20% of its original volume, or its volume < 10 to 15 mL. CT scans are performed every 24 hours for the duration of treatment with thrombolytic agents and then 24 hours after the final administration.

39.6.1 Injecting Recombinant Tissue Plasminogen Activator

In preparing to inject a thrombolytic agent (rt-PA or urokinase) in a sterile manner, the surgeon washes his or her hands, puts on a surgical mask, and uses sterile gloves. A sterile drape is placed on

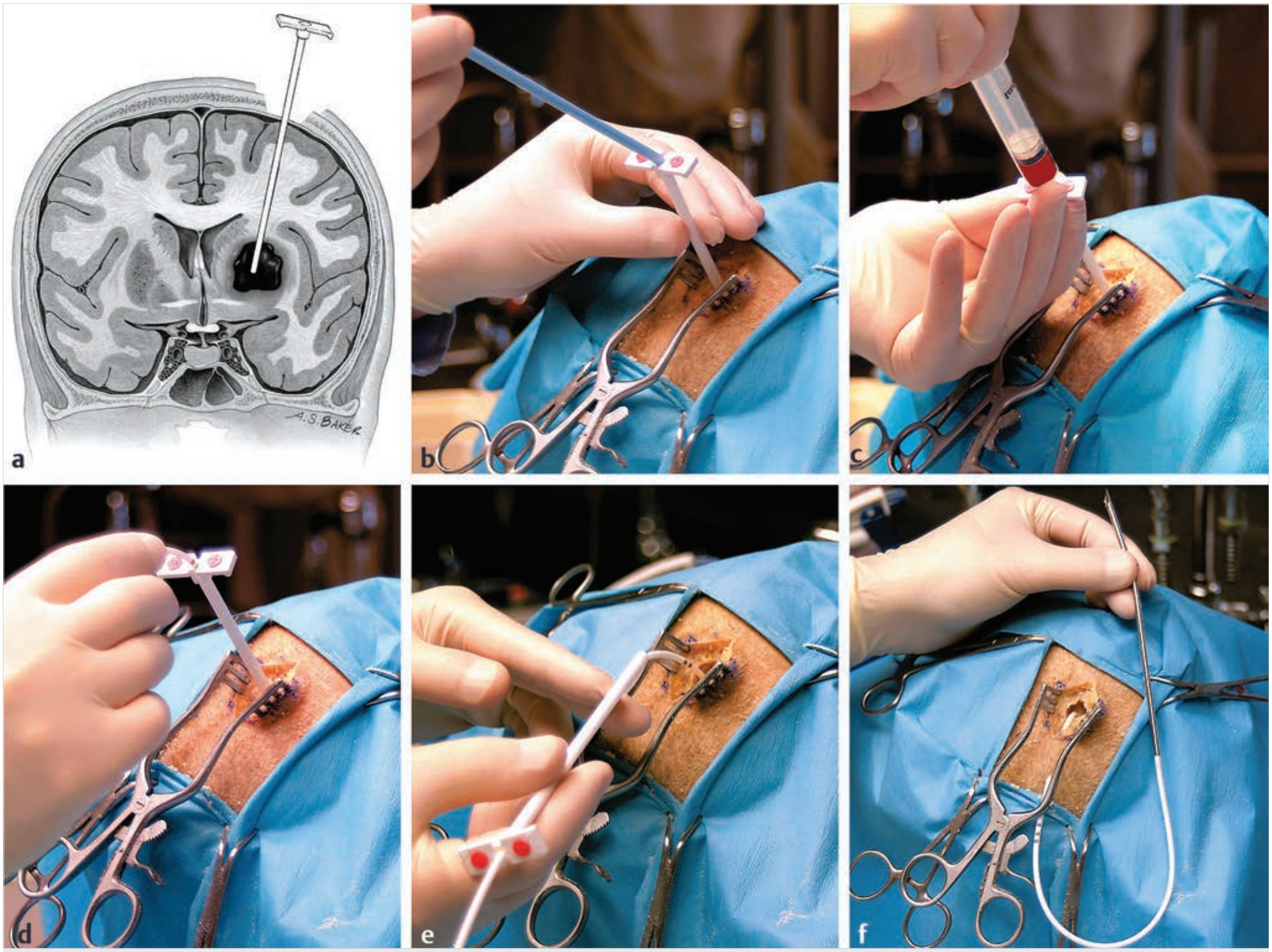

Fig. 39.1 Stepwise technique for minimally invasive surgical evacuation of a clot after intracerebral hemorrhage in the supratentorial compartment. **(a)** After making a large burr hole over the largest part of the hematoma, the surgeon makes about 1-cm incision to open the dura. In one pass, a 14 French peel-away introducer, placed into the central core of the hematoma, is inserted to a depth at least two thirds of overall hematoma diameter. **(b-f)** Cadaveric photographs. **(b)** Inner stylet is carefully removed while the cannula remains within the clot. **(c)** Using a 10-mL syringe, hematoma is aspirated until there is no longer a fluid component of the clot. **(d)** Soft catheter is passed through the cannula into the residual hematoma cavity. **(e)** Cannula is removed while the soft catheter remains. **(f)** Catheter is tunneled away from the incision by a stab incision, fixed to the skin, and connected to a closed drainage bag system (From the Mayfield Clinic reprinted with permission).

the field. The stopcock site is cleaned with povidone iodine (Purdue Frederick Co., Stamford, CT) and/or alcohol and allowed to dry. After injection of the rt-PA via the intracranial catheter (maximum rate 1 mL/min), the catheter is flushed with 2 to 3 mL of preservative-free normal saline. The soft catheter drainage system is closed for 60 minutes and then reopened at the level of the head until the next scheduled dose. There is no need for aspiration any time after surgery, and all drainage should be achieved by gravity only.

39.6.2 Catheter Removal

Administration of the thrombolytic agent is repeated until the hematoma volume is reduced to 20% of the initial clot volume or volume < 10 to 15 mL. Thrombolytic agents have been used for 72 hours, or significant rebleeding as noted on CT as part of the daily follow-up, or prompted by clinical deterioration. If rebleeding is noted, thrombolytic agents should be discontinued (▶ Fig. 39.2**c**). We suggest that the intracranial catheter be removed at least 3 hours, but preferably 12 to 24 hours, after the last thrombolytic injection. The catheter tip is sent to bacteriology for culturing and the skin is then sutured.

39.6.3 Complications

MIS evacuation of intracerebral hemorrhage can effectively reduce clot burden with minimal disruption of viable tissue. The main risk of intracranial thrombolytic therapy, the precipitation of rebleeding, is managed by discontinuing the thrombolytic agent. Large clots associated with neurological deterioration can be considered for open surgical evacuation, if indicated. Catheter malposition or dislodgement should prompt eventual repositioning or discontinuation of the treatment. Infection and seizures, though possible, are also infrequent complications.

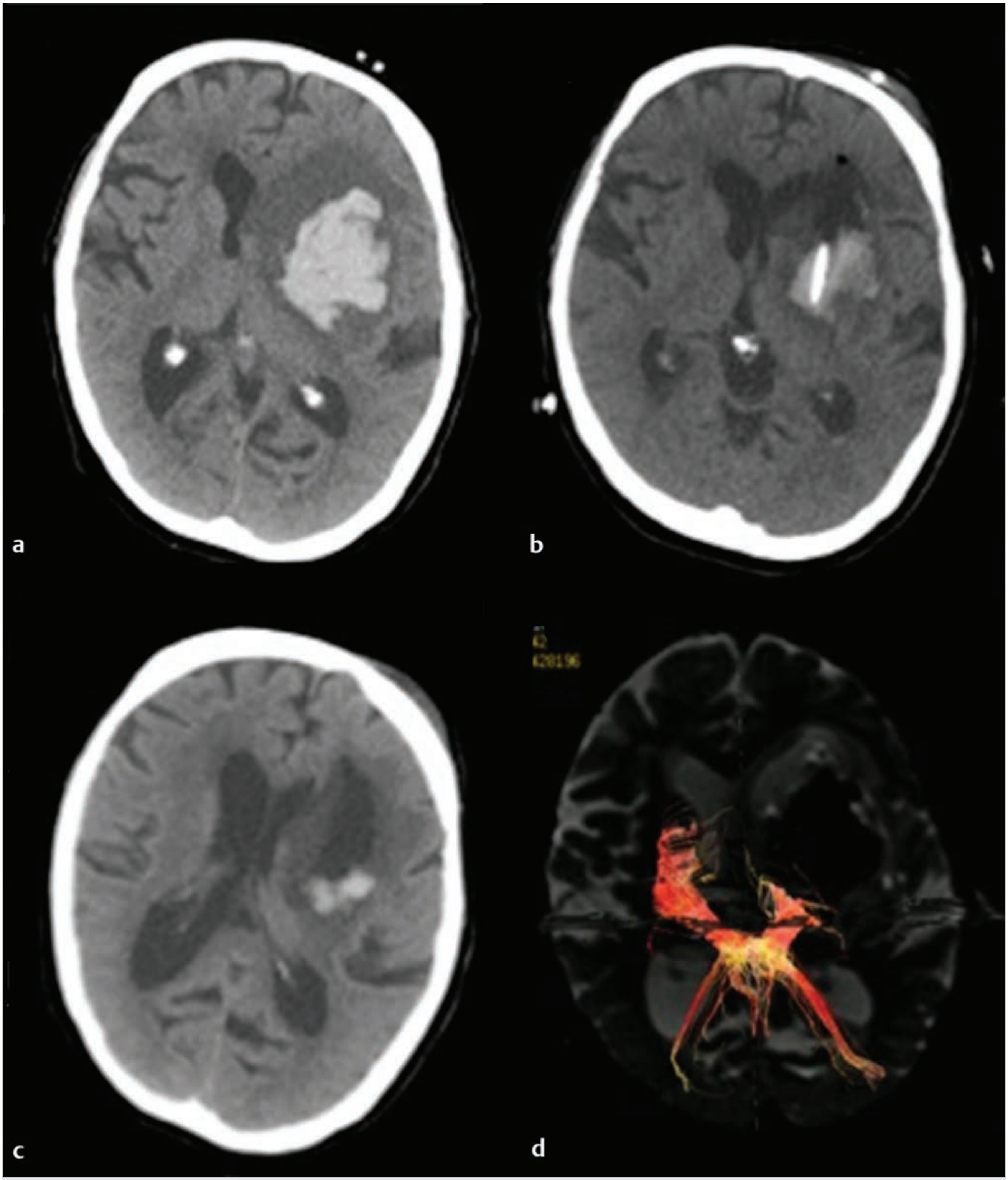

Fig. 39.2 Progression of intracerebral hemorrhage (ICH) before, during, and after minimally invasive surgical evacuation. **(a)** Preoperative computed tomography (CT) scans of a patient with a large left basal ganglia ICH. **(b)** Immediate postoperative CT of the head demonstrating catheter placement within the clot and significant amount of clot removed during the procedure. **(c)** Post-drainage CT demonstrating near complete evacuation of ICH. **(d)** Preoperative diffusion tensor imaging image of the bilateral corticospinal tracts in patient shows displacement and increased anisotropy of the ipsilateral corticospinal tract.

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